THESIS

FOR THE AWARD OF DEGREE OF

DOCTOR OF MEDICINE

SESSION 2022-2025

A COMPARITIVE STUDY OF CORD BLOOD LIPID PROFILE IN SMALL FOR GESTATIONAL AGE VS APPROPRIATE FOR GESTATIONAL AGE NEONATES

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ACKNOWLEDGEMENT

It is a matter of immense pride and honor to be a part of Santosh Medical College and Hospital, an institution that provided me with the necessary resources, supportive infrastructure, and the invaluable guidance of a dedicated and experienced faculty. This journey would not have been possible without the environment of academic excellence and encouragement fostered here.

First and foremost, I extend my deepest gratitude to **Dr. Alka Agrawal**, my respected guide, for her unwavering support, invaluable guidance, and continuous encouragement throughout the course of this thesis. More than a guide, she has been a true mentor and role model. Her profound knowledge and insight have been instrumental in shaping the direction of this research. Beyond this thesis, her mentorship has provided me with academic direction and has significantly contributed to my growth—not just as a doctor, but as a person.

I am equally thankful to my co-guide**, Dr. Radhika Mantry**, whose constructive feedback and persistent motivation have helped refine and elevate my work. Her support has been a constant source of strength through every phase of this journey.

My deepest and most heartfelt gratitude goes to my parents and sibling ,to whom I owe everything **Mr. Ravinder Singh Mrs. Harjeet Kaur and Mr. Sifat Singh Bhatia** . Without them, none of this would have been possible. They have been my greatest support system, my first teachers, and the guiding light through every storm. Their unconditional love, endless sacrifices, and unwavering belief in me have been the driving forces behind everything I have achieved. Their presence in my life continues to inspire me to be a better human being every day.

I would be remiss not to express my heartfelt gratitude to my beloved dogs — **Olive and Goofy**. I also want to remember my dearest **Pluto**, who now watches over me from heaven. He was the love of my life, and I believe with all my heart that he sent these two little souls to watch over me and fill my days with love and laughter

Olive and Goofy — who are truly like my children. Adopted at the very beginning of my residency, they have been my unwavering companions, bringing boundless joy and warmth to even the most difficult days. Their unconditional love and unspoken empathy have the incredible ability to lift my spirits, no matter how heavy the day has been. They are the light of my life, and the comfort and happiness they bring are beyond words. Even without the ability to speak, they have shown me the deepest form of communication — one of

pure love, loyalty, and healing.

To my dearest friends who are more like family — **Dr. Jasleen Kaur, Dr. Aashita Malik, Dr. Aahna Singh, , Dr. Kanwardeep Singh and Dr Raj** — thank you for always being just a call away. Your constant support, heartfelt encouragement, and ability to listen, understand, and uplift me even in silence has brought immense joy and comfort throughout this journey. You have been my strength in more ways than I can express.

I am sincerely grateful to my **Head of Department, Dr. Urmila Jhamb**, for her constant support and for being a remarkable leader. Her dedication to the department and to teaching has always inspired me. Her commitment to excellence has had a profound influence on my learning experience.

I would also like to express my deep admiration and heartfelt thanks to **Dr. D.K. Jha and Dr. Mohit Sharma.** They have been more than just exemplary teachers—they have been true inspirations. Their clinical acumen, compassionate care, and the way they carry themselves as professionals are qualities I deeply admire and aspire to emulate as I grow in my journey as a doctor.

I extend my gratitude to my consultants—**Dr. Veenu Agarwal, Dr. Abhishek, Dr. Rashi, Dr. Anisa and Dr Aarti** —for their constant guidance, patience, and support. Each of them has played an essential role in enriching my learning experience during my residency.

A special mention to my flatmate, **Dr. Sumedha**, whose constant presence, understanding, and support have been a true blessing. Living together, she has been more than just a friend—her companionship brought comfort during stressful times and joy in everyday moments. I would also like to express my sincere appreciation to my fellow postgraduates and friends from the hospital **, Dr. Vibhu, Dr. Hashim, and Dr. Shubhangi** — for your camaraderie, support, and for making even the most challenging days of residency lighter and more memorable. Your presence turned routine days into cherished moments.

I would also like to express my heartfelt gratitude to **Head Sister Shijita James** , whose warmth, kindness, and infectious laughter made even the toughest days brighter. Her care and thoughtful presence have been a source of both comfort and strength throughout my time here.

Lastly, I am deeply thankful to all my **seniors, juniors, and the patients who participated in the study**. Their cooperation and involvement were integral to the successful completion of this research.

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LIST OF ABBREVIATIONS

1. AGA	Appropriate for Gestational Age
2. SGA	Small for Gestational Age
3. LBW	Low Birth Weight
4. VLBW	Very Low Birth Weight
5. ELBW	Extremely Low Birth Weight
6. NICU	Neonatal Intensive Care Unit
7. HDL	High-Density Lipoprotein
8. LDL	Low-Density Lipoprotein
9. VLDL	Very Low-Density Lipoprotein
10. TC	Total Cholesterol
11. TG	Triglycerides
12. BMI	Body Mass Index
13. PI	Ponderal Index
14. GA	Gestational Age
15. LGA	Large for Gestational Age
16. SD	Standard Deviation
17. CI	Confidence Interval
18. OR	Odds Ratio
19. RR	Relative Risk
20. IUGR	Intrauterine Growth Restriction
21. NEC	Necrotizing Enterocolitis
22. ROP	Retinopathy of Prematurity
23. CPAP	Continuous Positive Airway Pressure
24. G2	Gravida 2 (Second Pregnancy)

25. G3	Gravida 3 (Third Pregnancy)
26. G4	Gravida 4 (Fourth Pregnancy)
27. ANC Visit	Antenatal Care Visit
28. HTN	Hypertension
29. DM	Diabetes Mellitus
30. CHD	Congenital Heart Disease / Coronary Heart Disease (depending on context)
31. LMP	Last Menstrual Period
32. EDD	Estimated Due Date
33. UTI	Urinary Tract Infection
34. RHD	Rheumatic Heart Disease
35. HC	Head Circumference
36. NBR	Neonatal Birth Record

INTRODUCTION

INTRODUCTION

Cord blood lipid profile analysis has garnered significant attention in neonatology and perinatal research due to its implications for understanding fetal development, metabolism, and neonatal outcomes. Lipids play an essential role in cellular structure and function, energy storage, and signaling pathways. The lipid profile of cord blood, which includes parameters such as total cholesterol (TC), triglycerides (TG), low-density lipoprotein cholesterol (LDL-C), and high-density lipoprotein cholesterol (HDL-C), provides critical insights into the intrauterine environment and its impact on the neonate. These factors are especially relevant for small for gestational age (SGA) neonates, a subgroup of newborns with unique metabolic and developmental challenges.[1]

SGA refers to neonates whose birth weight is below the 10th percentile for their gestational age. These infants experience suboptimal intrauterine growth, often due to maternal, placental, or fetal factors. Conditions such as maternal hypertension, placental insufficiency, and genetic abnormalities can restrict nutrient and oxygen supply, thereby impairing fetal growth. The altered intrauterine milieu in SGA pregnancies is associated with a spectrum of metabolic adaptations, including changes in lipid metabolism.

SGA neonates are at an increased risk of developing metabolic syndrome later in life, including insulin resistance, dyslipidemia, and cardiovascular diseases. Barker et al.'s "fetal origins of adult disease" hypothesis emphasizes the link between low birth weight and adult-onset chronic conditions, underscoring the importance of studying lipid profiles in this population [2]. Lipids serve as vital energy sources and structural components for fetal growth and development, particularly in the central nervous system. Abnormal lipid metabolism during intrauterine life in SGA neonates may have lasting repercussions.

Cord blood, obtained from the umbilical vein and arteries at birth, reflects the metabolic and nutritional status of the fetus. The lipid profile of cord blood includes total cholesterol (TC), a precursor for steroid hormone synthesis and a component of cell membranes; triglycerides (TG), which serve as major energy reservoirs stored in

adipocytes; low-density lipoprotein cholesterol (LDL-C), which transports cholesterol to peripheral tissues and is often referred to as “bad cholesterol”; and high-density lipoprotein cholesterol (HDL-C), which facilitates reverse cholesterol transport to the liver and is termed “good cholesterol.” The cord blood lipid profile reflects intrauterine lipid metabolism and placental lipid transfer. Maternal lipids, especially during the third trimester, contribute significantly to fetal lipid levels. However, the placental barrier modulates this transfer, influencing fetal lipid concentrations [3]. Understanding these mechanisms is particularly crucial for SGA neonates, who often experience altered placental function.

SGA neonates often exhibit deviations in their lipid profile compared to appropriate for gestational age (AGA) counterparts. Studies have reported lower HDL-C and higher LDL-C levels in SGA neonates, indicative of an atherogenic profile [3]. Such abnormalities may arise from placental dysfunction, which impairs lipid transfer due to suboptimal placental perfusion; fetal adaptations, such as redistribution of blood flow to vital organs like the brain (brain-sparing effect) at the expense of adipose tissue development; and epigenetic changes, including alterations in gene expression regulating lipid metabolism due to intrauterine stress. These metabolic changes can persist postnatally, predisposing SGA individuals to cardiovascular and metabolic disorders in adulthood.[4]

Maternal factors significantly influence the cord blood lipid profile. Maternal hyperlipidemia during pregnancy, often observed in conditions like preeclampsia and gestational diabetes mellitus (GDM), alters the lipid supply to the fetus. Placental lipid metabolism, involving uptake, synthesis, and transfer, also plays a pivotal role. Studies have shown that placental lipid transport proteins such as fatty acid transport proteins (FATPs) and lipoprotein lipase (LPL) are dysregulated in pregnancies complicated by intrauterine growth restriction (IUGR), a condition closely related to SGA [5].

The lipid profile in cord blood serves as a biomarker for assessing fetal metabolic health. Key clinical implications include predicting neonatal outcomes, as abnormal lipid levels are associated with adverse outcomes such as hypoglycemia, hypothermia, and feeding difficulties in SGA neonates; assessing long-term risks, as dyslipidemia

in SGA neonates can indicate future risks for obesity, type 2 diabetes, and cardiovascular diseases; and guiding nutritional interventions, as understanding lipid metabolism can help optimize postnatal nutrition to prevent metabolic complications.[6-7]

Despite significant advancements, several gaps remain in our understanding of the cord blood lipid profile in SGA neonates. Longitudinal studies linking neonatal lipid profiles to long-term metabolic outcomes are limited. Additionally, there is a lack of standard reference ranges for cord blood lipid parameters in different populations and gestational ages. Exploring the role of genetic and epigenetic factors in lipid metabolism may provide deeper insights into the pathophysiology of SGA.

This study aims to compare the cord blood lipid profile of SGA neonates with AGA counterparts, evaluate the influence of maternal and placental factors on lipid level

NEED FOR THE STUDY

Literature on the topic in both preterm and term neonates is still lacking in Indian studies.

A combined study including both preterm and term neonates of small for gestational age and appropriate for gestational age neonates will help us to find out how prematurity and growth retardation during antenatal period can have an affect later stages life and lead to cardiovascular disorders .

By comparing the lipid profile in these neonates, we can establish if there is a need to counsel parents of such neonates starting early in perinatal period.

AIMS AND OBJECTIVES

AIMS & OBJECTIVES

Aim:

A Comparative study of cord blood lipid profile in small for gestational age vs appropriate for gestational age neonates

Objectives:

Primary Objective: To compare lipid profile parameters in term and preterm neonates

Secondary Objective: To identify relation of cord blood lipid profile in term and preterm , small for gestational age and appropriate for gestational age neonates and increase risk of atherosclerotic cardiovascular diseases later on in adulthood:

To compare the lipid profile in

- Term/preterm
- Small for gestational age/appropriate for gestational age
- Total cholesterol level
- Triglyceride level
- HDL
- VLDL / LDL

REVIEW OF LITERATURE

REVIEW OF LITERATURE

REVIEW OF LITERATURE

The umbilical cord provides the pathway for unhindered blood transport from the placenta to the foetus and vice versa. Aristotle (384–322 BC) originally identified the umbilical cord as the connection between the mother and unborn child. (33) The umbilical cord is considered both the physical and emotional attachment between mother and fetus. This structure allows for the transfer of oxygen and nutrients from the maternal circulation into fetal circulation while simultaneously removing waste products from fetal circulation to be eliminated maternally. On the other hand, mothers associate an emotional connection to the fetus through the cord. It may merit consideration as the route of love and care during pregnancy. Thus, some poets call it the string of life. [8]

- **ANATOMY OF UMBILICAL CORD:**

1. Embryology of Umbilical Cord: [9-10]

The primitive umbilical ring present between amnion and embryonic ectoderm later develops into UC. The structures passing through this ring are connecting stalk containing allantois, umbilical vessels, yolk sac with vitelline vessels. Yolk sac remains attached to midgut by a narrow omphalomesentric duct (yolk stalk). At 4th week during folding of embryo, amniotic cavity expands at the expense of chorionic cavity. Later amnion envelops the connecting stalk, yolk stalk, and allantois forming an epithelial covering for UC. Yolk sac and allantois are vestigial structures. Both are early sites for blood formation. Primordial germ cells also originate in the wall of yolk sac. Therefore, presence of yolk sac and allantois is essential for the normal embryonic development. Connecting stalk connects extra embryonic mesoderm to the embryonic disc. This mesoderm, later converts into gelatinous substance known as Wharton's jelly. As the development progresses the connecting stalk containing allantois, yolk stalk and umbilical vessels gets smaller in diameter and increases in length. Later, yolk stalk (vitelline duct) and allantois get obliterated.

Proximal part of allantois persists as urachus, which extends from bladder to

umbilical region and later it persists as median umbilical ligament. Only umbilical vessels remain surrounded by Wharton's jelly at the end of development. There are two UAs formed from allantois arteries. The paired umbilical arteries pass through connecting stalk and become continuous with vessels in chorion and embryonic part of placenta. They carry poorly oxygenated blood to placenta. Proximal part of UAs remains as internal iliac and superior vesical arteries. Distal part gets obliterated after birth and becomes medial umbilical ligament. [9]

Initially, two umbilical veins carry well-oxygenated blood from the chorion and run on each side of liver to sinus venosus. As liver develops, umbilical veins lose their connection with heart and end in liver. Right UV disappears during 7th week and only left UV persists. Large venous shunt, the ducts-venosus develops within liver and connects UV with inferior vena cava. Intra-abdominal part of UV becomes the round ligament, which runs from umbilicus to porta hepatis after birth.

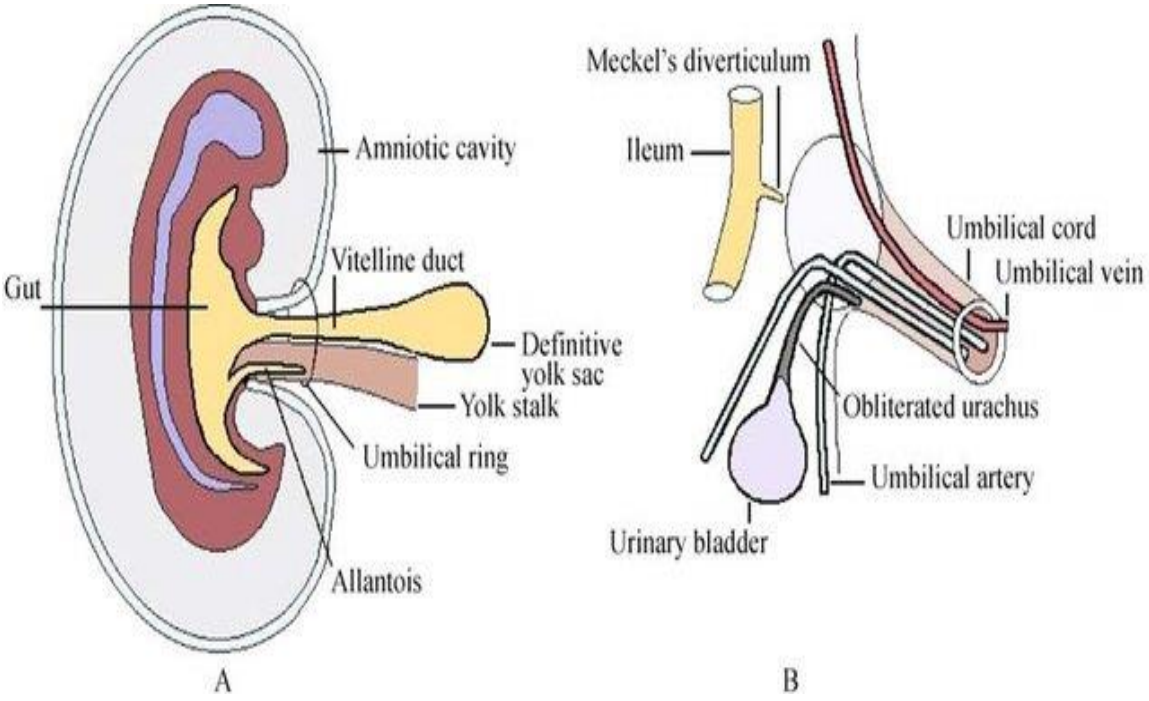


Fig. 1: Diagrams showing the (A) folding of the embryo and formation of the primitive umbilical cord and (B) definitive umbilical cord. [11]

2. Normal Structure of Umbilical Cord:

UC is 2cm in diameter and 50-60 cm long flexible cord. If it is greater than 70cm and less than 35cm then it is called as long and short cord respectively. UAs coil around single UV in left side or anticlockwise twist, which is commonly seen. Mean coiling index (dividing the number of complete vascular coils by the total length of UC in cm) is 0.26±0.09 coils/cm.90 Umbilical vessels are longer than cord, so they are tortuous and frequently form nodulation on surfaces or false knots, which are essentially varices. Transverse section of a normal UC consists of two umbilical arteries and single umbilical vein surrounded by a connective tissue WJ. Single layer of cuboidal to columnar cells of amnion lines these structures. Amnion layer maintains the fluid pressure of cord. As per the standard histological textbook and literature, based on the diameter of vessel, umbilical vessels look like a medium-sized vessel. [12-13] Umbilical vessels are different in structure and function as compared to systemic vessels in the body. Umbilical vessels have well defined tunica intima and tunica media, whereas tunic adventitia is not clear. WJ performs the function of adventitial layer and gives protection to the umbilical vessels. Normally medium sized artery has internal elastic lamina as a characteristic feature. Whereas umbilical artery does not possess internal elastic lamina (IEL), instead UV has IEL. Another unique feature of umbilical vessel is they devoid of vasa vasorum and innervation.

3. Microscopic Structure of Umbilical Vein (UV):

Electron microscopic and quantitative studies of microstructure of UV revealed the following histomorphological features. [14]

Tunica Intima: It consists of single layer of endothelial cells, subendothelial layer and IEL. IEL is integrated, connected to underlying SMCs by few gaps and separates the tunica intima from tunica media.

Tunica Media: Smooth muscle forms the structural component of media, which is thinner than media of artery. Few layers of SMCs arranged longitudinally inside followed by an outer predominant circular pattern. [15] SMCs are long, spindle in shape with centrally situated nucleus. Collagen and few elastic fibres are present between the SMCs. Sexton et al reported that as the gestational age increases,

thickness of the media and size of lumen also increases. [14] He mentioned that at 13 weeks layers of SMCs in UV were 12-14 cells thick, which increased to 24-28 cells thick by 16 weeks. Li WC et al reported 14-23 layers of SMC and term, diameter of UV is 2 to 3mm. [15] Therefore, UV is thicker than medium sized vein and similar to small calibre arteries like brachial, ulnar, radial, coronary and tibial arteries. UV has abundant SM, moderate amount of collagen and elastin, so it shows good expansion and contraction capacity. Media was indistinctly separated from adventitial layer.

Tunica adventitia: This layer is thinner than tunica media. It is ill-defined and composed of some amount of collagen and few SMCs. External elastic lamina was not distinct and continuous. Vasa vasorum and nerves are absent. [16]

4. Microscopic Structure of Umbilical Artery (UA):

Electron microscopic studies have evaluated the normal histomorphological features of UA as follows; [14, 17,18]

Tunica Intima: Is the innermost layer and is lined by endothelial cells. Subendothelial area consists of small amount of ground substance, few SMCs and rich in bundles of collagen fibres. IEL is absent. Hence, demarcation between intima and media is not distinct.

Tunica Media: Smooth muscle is the main component of media. It showed distinct inner and outer layers. Inner layer of media: Here SMCs are irregular and loosely arranged. Few SMCs are embedded in rich ground substance along with fine, sparse elastic fibres and network of reticular fibres. SMCs are undifferentiated and have less cytoplasm with poorly defined outlines and nuclei are obliquely situated.

Outer layer of media: Here SMCs are tightly packed, parallel to each other and arranged in circular pattern that is perpendicular to the direction of blood stream. SMCs are well differentiated with long spindle shaped cells. Sexton et al demonstrated that along with gestational age SMCs attained contractile phenotype structure. [14] Thickness of media increased from 19-21 cells to 27-45 cells thick with increasing gestational age. Raffino et al demonstrated that SMCs in UA progressively acquire undifferentiated to fully differentiated contractile phenotype during third

trimester. [19] Contact between adjacent SMCs is high and gap junctions are not observed. External elastic lamina is not clearly visible.

Tunica Adventitia: It consists of sparse connective tissue with few scattered SMCs and not well appreciated from media. No vasa vasorum and nerves are seen. UA has thick muscular tunic that eliminates the need of IEL. Presence of thick SM enables it to contract or relax according to hemodynamic state. Therefore, UA functions as conducting tube and also act as regulating organ for blood flow. Whereas, compared to regular vein UV cannot stretch or retract due to presence of IEL, which prevents overstretching. Because of IEL, it is able to contract and behaves as artery.[14]

Supply of Nutrition: Hoang-Ngoc et al conducted a study to observe the ultrastructure and function of the fetal umbilical vessels. Since the umbilical vessels are devoid of vasa vasorum, they get their nutrition from blood flowing through the lumen itself. This active diffusion process was known as "plasma perfusion." Discontinuous endothelial basement membrane, fenestrated internal elastic membrane and loosely arranged SMCs in inner layer of media enable the substance to diffuse from endothelial cells to reach muscular tunic. [20]

- **SGA (SMALL FOR GESTATIONAL AGE):**

Two neonatal problems may influence both birthweight and neonatal mortality risk: preterm delivery and acute growth retardation. Infants that are delivered before 37 completed weeks (259 days) of pregnancy are classified as preterm births. They tend to be lighter than term births, although a baby can be preterm. The definition and significance of growth retardation is confused in much clinical and epidemiological literature. This is partly because of the assumption that small infants must be growth retarded. There are various definitions of growth retardation currently in use: for example, birthweight less than the fifth centile of standard weight for gestational age; birthweight less than 2.5 kg in a term infant; or birthweight less than 2 standard deviations below the population mean. [21] These babies are often described as small for gestational age (SGA). In view of the increased mortality seen in low birthweight groups, it is a small conceptual leap to regard SGA babies as being the result of a

disease process that has caused them to be growth retarded. This thinking equates SGA with intrauterine growth retardation (IUGR). These terms are often used interchangeably in the literature.

The term' small for gestational age' represents a statistical group of infants whose weight and/or crown-heel length is less than expected for their gestational age and sex. [22] The definition of SGA is not straightforward as it requires accurate knowledge of gestational age, measurements of birth weight, length, and head circumference, and a reference data from a relevant population. The cut-off has been variably set at the 10th centile, 3rd centile, or at less than –2 SD from the mean (approximately 2nd centile). [23] The International Small for Gestational Age Advisory Board Consensus Development Conference Statement 2001, and the Consensus Statement of the International Societies of Pediatric Endocrinology and the Growth Hormone Research Society 2007, recommend that SGA should be defined as a neonate whose birth weight or birth crown-heel length is at least 2 standard deviations (-2 SD) below the mean for the gestational age, based on data derived from a reference population. [23-24]

These babies can be subclassified into SGA for weight, length, or for both weight and length.[24] SGA must be differentiated from low birth weight and intrauterine growth retardation (IUGR). The term SGA refers not to fetal growth but to the size of the infant at birth whereas, the term IUGR suggests diminished growth velocity in the fetus occurring in utero, as documented by at least two intrauterine growth assessments. A child who is born SGA has not necessarily suffered from IUGR, and infants who are born after a period of IUGR are not necessarily SGA. [25]

Epidemiology:

The data on the incidence of SGA births are scarce in many countries because birth length and gestational age are rarely recorded in National databases. Based on the available data, it has been estimated that between 2.3 and 10% of all infants are born SGA. [26-27] India has a high incidence of low birth weight (LBW) and SGA babies [26-27]. The incidence of LBW in India is about 30% babies in contrast to 5-7% in developed countries. [27] A large percentage (approximately 70%) of LBW are SGA [26,27]]. Kushwaha, et al. [28] studied 750 hospital deliveries (term singleton

neonates) and found that 28.4% were SGA, which is almost similar to the incidence of 25% reported by Mehta, et al. [29] Thus, there is a huge burden of LBW and SGA in our country which needs to be addressed.

The causation of SGA is multifactorial. Fetal factors include chromosome abnormalities and genetic defects. Maternal factors involve age, weight and height, parity, chronic diseases, infections, impairment of nutritional status, and substance abuse. Placental factors include structural abnormalities and insufficient perfusion. Thus, the ability to reach an optimal birth weight results from the interaction between the fetal growth potential (the fetal factors) and the environment (placental and maternal factors). [23] The definition of SGA does not take into account the background growth-modifying factors such as maternal size, ethnicity, and parity. These factors may help in understanding the mechanisms and implications of being born SGA. Narang, et al. in 1997 concluded that idiopathic intrauterine growth retardation is the commonest cause of SGA in Indian babies, followed by pregnancy induced hypertension which is one of the most important risk factors for SGA/IUGR. [30]

1. **Etiology of SGA Infant:**

Causes may be divided into those in which the growth restriction is:

- **Symmetric:** Height, weight, and head circumference are about equally affected.
- **Asymmetric:** Weight is most affected, with a relative sparing of growth of the brain, cranium, and long bones.

Symmetric growth restriction usually results from a fetal problem that begins early in gestation, often during the 1st trimester. When the cause begins relatively early in gestation, the entire body is affected, resulting in fewer cells of all types. Common causes include

i. Genetic disorders.[31]

ii. First-trimester congenital infections (e.g., with cytomegalovirus, rubella virus, or Toxoplasma gondii)

Asymmetric growth restriction usually results from placental or maternal problems that typically manifest in the late 2nd or the 3rd trimester. When the cause begins relatively late in gestation, organs and tissues are not equally affected, resulting in asymmetric growth restriction. Common causes include[31]

i. Placental insufficiency resulting from maternal disease involving the small blood vessels (e.g., preeclampsia, hypertension, renal disease, antiphospholipid antibody syndrome, long-standing diabetes)

ii. Relative placental insufficiency caused by multiple gestation

iii. Placental involution accompanying post maturity

iv. Chronic maternal hypoxemia caused by pulmonary or cardiac disease

v. Maternal malnutrition

vi. Conception using assisted reproductive technology

An infant may also have asymmetric growth restriction and be small for gestational age (SGA) if the mother is a heavy user of opioids, cocaine, alcohol, and/or tobacco during pregnancy. [32-33]

2. Pathophysiology:

There is a compromise in the supply of nutrients reaching the fetus in FGR SGA. In an attempt to maximize the survival chance, the fetus responds to the reduction in its nutrient supply by reducing its overall size but preserving certain functions like brain growth, lung maturation, and increasing red blood cell production. [34] The fetus prioritizes blood supply to more vital organs such as the brain, heart, adrenals, and placenta. There is a decrease in the total body fat, lean mass, and mineral contents in infants with severe FGR SGA, giving the neonate a wasted appearance. [35]

FGR SGA is divided into symmetric and asymmetric growth restriction. Infants with symmetric FGR SGA have a proportionate reduction in all organ

systems, and the growth restriction typically begins early in the gestation. The incidence of symmetric FGR SGA ranges from 38% to 45%, and factors such as first-trimester congenital infection and chromosomal abnormalities have been implicated. In asymmetric FGR SGA, which constitutes about 55% to 61% of FGR SGA cases, the growth in head circumference is preserved while the length and weight are affected. [36-38]

The growth restriction in asymmetric FGR SGA begins in the late 2nd or 3rd trimesters and usually results from a reduction in fetal nutrients due to placental or maternal factors. Continued brain growth is prioritized. Ponderal index (PI), which is a ratio of body weight to length (PI=[weight (g) x 100]/[length (cm)]), is an important tool to detect FGR SGA, especially in infants with asymmetric FGR SGA. [39]

3. Diagnosis and Investigation of SGA Babies:

i. Optimum method of diagnosing an SGA fetus:

Fetal abdominal circumference (AC) or estimated fetal weight (EFW) < 10th centile can be used to diagnose an SGA fetus. Use of a customised fetal weight reference may improve prediction of an SGA neonate and adverse perinatal outcome. In women having serial assessment of fetal size, use of a customised fetal weight reference may improve the prediction of normal perinatal outcome. Routine measurement of fetal AC or EFW in the third trimester does not reduce the incidence of an SGA neonate nor does it improve perinatal outcome. Routine fetal biometry is thus not justified. Change in AC or EFW may improve the prediction of wasting at birth (neonatal morphometric indicators) and adverse perinatal outcome suggestive of FGR. When using two measurements of AC or EFW to estimate growth velocity, they should be at least 3 weeks apart to minimise false–positive rates for diagnosing FGR. More frequent measurements of fetal size may be appropriate where birth weight prediction is relevant outside of the context of diagnosing SGA/FGR. Where the fetal AC or EFW is < 10th centile or there is evidence of reduced growth velocity, women should be offered serial assessment of fetal size and umbilical artery Doppler.[39]

Neonatal Growth Curves

Neonatal growth curves are essential tools used to assess the growth and development of newborns, comparing their weight, length, and head circumference to standard reference populations. These curves help classify newborns as small for gestational age (SGA), appropriate for gestational age (AGA), or large for gestational age (LGA) and are crucial in identifying neonates at risk for metabolic and developmental complications.[40]

Types of Neonatal Growth Curves[41-42]

A. Fenton Growth Charts (Preterm Neonates)

- Developed specifically for **preterm infants (<37 weeks gestation)**.
- Based on a large dataset of preterm infants to provide accurate percentiles for weight, length, and head circumference.
- Helps monitor the growth of preterm babies and assess **catch-up growth** after birth.

B. WHO Growth Standards (Term Neonates)

- Used for **term neonates (≥37 weeks gestation)**.
- Derived from a **multinational study by the World Health Organization (WHO)**.
- Provides percentile curves for weight, length, and head circumference from **birth to 5 years**.

C. INTERGROWTH-21st Standards

- A global growth standard incorporating data from multiple countries.
- Includes **preterm and term neonates** and follows growth trends into infancy.
- Helps **compare growth patterns across different populations**.[42]

Interpretation of Growth [41-42]

Classification	Percentile Range
SGA (Small for Gestational Age)	<10th percentile
AGA (Appropriate for Gestational Age)	10th-90th percentile
LGA (Large for Gestational Age)	>90th percentile

- **SGA neonates** are at higher risk for **hypoglycemia, metabolic disorders, and growth failure**.
- **LGA neonates** are at risk for **birth complications, obesity, and metabolic syndrome** in later life.

Importance of Growth Curves in Neonatal Lipid Studies

Growth curves help correlate **birth weight, ponderal index, and lipid profile alterations** in SGA and preterm neonates. **Barker's Hypothesis** suggests that SGA neonates with **low birth weight** are at **higher risk for dyslipidemia and cardiovascular disease** later in life. Monitoring postnatal growth patterns helps identify infants at risk for **long-term metabolic syndrome**.[41]

Ponderal Index in Neonates

The ponderal index (PI) is a crucial anthropometric measurement used to assess neonatal nutritional status and fetal growth patterns. Unlike birth weight alone, the ponderal index provides insights into the proportionality of intrauterine growth by considering both weight and length. It helps distinguish between symmetrical and asymmetrical intrauterine growth restriction (IUGR) and serves as an important marker of fetal malnutrition.[43-44]

A low ponderal index (<2.2 kg/m^3) suggests intrauterine wasting, often seen in asymmetrical growth restriction, where the fetus experiences late gestation

malnutrition, leading to normal length but reduced weight. Conversely, a high ponderal index (>2.9 kg/m³) may indicate excessive intrauterine growth or macrosomia, commonly seen in infants of diabetic mothers. Appropriate ponderal index (2.2–2.9 kg/m³) indicates proportionate fetal growth.[45]

Clinical Significance of Ponderal Index in Neonatal Metabolic Health

Neonates with low ponderal index often show metabolic disturbances, including altered lipid profiles, increased insulin resistance, and higher risks of developing metabolic syndrome, hypertension, and cardiovascular diseases in later life. Several studies suggest that SGA neonates with low ponderal index have higher triglyceride levels and lower HDL levels, reflecting an altered fetal lipid metabolism due to intrauterine nutritional deprivation.[46]

The relationship between PI and cord blood lipid profile has been studied, showing that low PI neonates often exhibit lipid abnormalities similar to those observed in SGA infants, reinforcing the importance of fetal programming in metabolic health.

Formulas to Calculate Ponderal Index[47]

Formula Name	**Formula**	**Interpretation**
Standard Ponderal Index	PI = (Birth weight in grams) / (Length in cm)³ × 100	Most commonly used; assesses fetal proportionality
Rohrer's Index	(Weight in kg) / (Height in m³)	Used in both neonates and adults
Corpulence Index	(Weight in kg) / (Length in cm)²	Alternative method for assessing growth patterns
BMI in Neonates	(Weight in kg) / (Length in m²)	Modified use in neonates but not as common as PI

The ponderal index provides a more accurate assessment of intrauterine growth than birth weight alone and should be considered alongside other neonatal parameters to predict long-term metabolic and cardiovascular outcomes.

Table 1: Table of available tests for prediction of restriction of fetal growth and compromise of fetal wellbeing.

Tests	Tests for restriction of fetal growth	Tests for compromise of wellbeing
History and Examination	◦ Clinical risk scoring ◦ Palpation of abdomen to assess size ◦ Symphyseal fundal height (SFH) measurement	◦ Clinical risk scoring ◦ Fetal movement counting
Ultrasound	**Biometry (anthropometric measures)** ◦ Single measures: Abdominal circumference (AC), Head circumference (HC), Biparietal diameter (BPD), Femur length (FL), Thoracic diameter, Abdominal diameter, Abdominal area, Chest area, Liver size, Thigh circumference, Subcutaneous fat ◦ Ratio measures: FL/AC, HC/AC, FL/HC, Head area/abdominal area, FL/ thigh circumference ◦ Composite measures: Estimated fetal weight (EFW), Fetal ponderal index, Total intrauterine volume, Trunk area x Crown Rump Length, ◦ Growth velocity measurements **Doppler** ◦ Uterine artery ◦ Umbilical artery ◦ Middle cerebral artery ◦ Descending aorta ◦ Internal carotid artery **Other** ◦ Amniotic fluid volume ◦ Placental grade	**Doppler** ◦ Uterine artery ◦ Umbilical artery ◦ Middle cerebral artery ◦ Venous Doppler ◦ Uteroplacental **Other** ◦ Amniotic fluid volume ◦ Biophysical profile
Biochemical and Haematological	◦ Oestriols, Human placental lactogen, Plasma fibronectin, Alpha Feto Protein, Human chorionic gonadotrophin, Beta-1 glycoprotein, Placental protein 10, Pregnancy Associated Plasma Protein A, dehydroepiandrosterone sulphate loading test, Epidermal growth factor, Amniotic fluid C-amino peptide, Serum cystine aminopeptidase, Schwangerschafts protein 1, Serum alpha 2 – macroglobulin, Maternal leukocyte zinc level, Form stability index	
Other tests	◦ Customised growth charts of SFH and ultrasound EFW	◦ Cardiotocography (CTG) ◦ Fetal ECG ◦ Fetal magnetocardiography

Ultrasound biometry:

Two systematic reviews have assessed the accuracy of ultrasound biometric measures, both as individual measures, as ratios, and combined (as the EFW). [48] Use of the 10th centile had better sensitivities and specificities than other commonly used centiles. In a low-risk population sensitivity varies from 0–10% and specificity 66–99% for any parameter. In a high-risk population, fetal AC < 10th centile had sensitivity ranging from 72.9–94.5% and specificity 50.6–83.8%. For EFW < 10th centile, sensitivity was 33.3–89.2% and specificity 53.7–90.9%. Meta–analysis was not performed in these systematic reviews due to the considerable clinical and methodological heterogeneity within the included papers. The potential advantage of EFW is that customised standards exist and accuracy can more easily be determined against birthweight.

A retrospective study has shown that among high-risk patients, EFW and AC < 10th centile within 21 days of delivery better predicted an SGA infant than AC < 10th centile (80% versus 49%, OR 4.26, 95% CI 1.94–9.16). [49] Adverse perinatal outcome was also highest when both measures were < 10th centile. Kayem et al found that measurement of AC in low-risk women at term was a better predictor of birth weight ≤ 2.5 kg than a single measurement of SFH (LR+ 9.9 versus 7.1, LR– 0.5 versus 0.6). [50]

Several studies have compared various formulae for estimating fetal weight in unselected patients. A prospective study compared 35 different formulae and found that most are relatively accurate at predicting birth weight up to 3500 g. [51] Another study found the Shepard and Aoki formulae to have the best intraclass correlation coefficient, with EFW showing the smallest mean difference from actual birth weight. [52] Although formulae have been developed for SGA fetuses, there is little evidence that accurate prediction of weight is substantially improved and, in this population, the Hadlock formula may be most appropriate to use. [53-55]

There is no evidence to recommend one specific method of measuring AC (directly or derived from abdominal diameters) nor which centile chart to use. The centile charts produced by Chitty et al was optimally constructed and are widely used. [56] The same maternal characteristics (maternal height, weight, parity and ethnic

group) that affect birth weight affect fetal biometric measures and fetal weight gain providing a rationale for the use of a customised AC or EFW chart. [57-58] A customised EFW < 10th centile is predictive of an SGA neonate (sensitivity 68%, specificity 89%). [59] Use of customised fetal weight centiles to define SGA has also been shown to improve the prediction of adverse prenatal outcome; [59-60] OR of adverse outcomes (stillbirths, neonatal deaths, referral to higher level or special care unit or Apgar score < 7 at 5 minutes) for SGA neonates versus those not SGA was 1.59 (95% CI 1.53–1.66) for the non–customised fetal weight reference compared with 2.84 (95% CI 2.71–2.99) for the customised reference. [59] Prediction of perinatal mortality was also improved by the customised reference (OR 3.65, 95% CI 3.40–3.92 versus OR 1.77, 95% CI 1.65–1.89). [60] A further study demonstrated that individual growth trajectories of low risk fetuses with normal outcome were less likely to cross below the 10th centile for fetal weight when using customised reference standards than when unadjusted standards were used. [61] However, no trials were identified that compared customised with non–customised EFW chartsp.

Biophysical tests:

Biophysical tests, including amniotic fluid volume, cardiotocography (CTG) and biophysical scoring are poor at diagnosing a small or growth restricted fetus. [62-63] A systematic review of the accuracy of umbilical artery Doppler in a high–risk population to diagnose a SGA neonate has shown moderate accuracy (LR+ 3.76, 95% CI 2.96–4.76; LR– 0.52, 95% CI 0.45–0.61). [64]

ii. Investigations indicated in SGA fetuses:

Offer a referral for a detailed fetal anatomical survey and uterine artery Doppler by a fetal medicine specialist if severe SGA is identified at the 18–20-week scan. Karyotyping should be offered in severely SGA fetuses with structural anomalies and in those detected before 23 weeks of gestation, especially if uterine artery Doppler is normal. Serological screening for congenital cytomegalovirus (CMV) and toxoplasmosis infection should be offered in severe SGA. Testing for syphilis and malaria should be considered in high-risk populations. Uterine artery Doppler has limited accuracy to predict adverse outcome in SGA fetuses diagnosed during the third trimester.

In severe SGA, the incidence of chromosomal abnormalities has been reported to be as high as 19%. [64] Triploidy was the most common chromosomal defect in fetuses referred before 26 weeks of gestation and trisomy 18 in those referred thereafter. Within this population, the risk of aneuploidy was found to be higher in fetuses with a structural abnormality, a normal amniotic fluid volume, a higher head circumference/AC ratio or a normal uterine artery Doppler. [65-66] One small study suggested that, in severely SGA fetuses, the rate of aneuploidy was 20% in fetuses presenting before 23 weeks of gestation, irrespective of the presence of structural anomalies, compared with 0% in fetuses presenting between 23–29 weeks of gestation. [67]

Fetal infections are responsible for up to 5% of SGA fetuses. [68] The most common pathogens are reported to be cytomegalovirus (CMV), toxoplasmosis, malaria and syphilis, although a recent multicentre study found no association between congenital toxoplasmosis and incidence of an SGA infant. [69] Malaria is a significant cause of preterm birth and LBW worldwide and it should be considered in those from, or who have travelled in, endemic areas. [70]

Normal Lipid Metabolism in Pregnancy

To understand the alterations in lipid profiles observed in SGA infants, it is important to first consider the normal changes in lipid metabolism that occur during pregnancy. Pregnancy is associated with significant changes in maternal lipid homeostasis, characterized by a progressive increase in all lipid fractions, particularly in the second and third trimesters.[71]

In early pregnancy, there is an increase in maternal fat accumulation, driven by enhanced lipogenesis and reduced lipolysis. This anabolic phase is thought to be mediated by the actions of progesterone and insulin, which promote the storage of energy reserves to support fetal growth in later pregnancy [71].

As pregnancy progresses, there is a shift towards a catabolic state, with increased lipolysis and fatty acid mobilization from maternal adipose tissue [9]. This shift is mediated by the actions of placental hormones such as placental lactogen and growth hormone, which promote insulin resistance and enhance the availability of lipid

substrates for fetal growth [72].

The placenta plays a crucial role in the transfer of lipids from the maternal circulation to the fetus. Lipids are transported across the placenta via several mechanisms, including passive diffusion, facilitated diffusion, and active transport . The main lipids transferred to the fetus are free fatty acids, which are derived from the hydrolysis of maternal triglycerides by placental lipases [72].

Fetal lipid metabolism is characterized by a high rate of de novo lipogenesis, which allows the fetus to synthesize its own lipids from glucose and amino acid precursors. The fetal liver is the primary site of lipid synthesis, with the majority of synthesized lipids being used for the growth and development of fetal tissues, particularly the brain [73].

Components of Cord Blood Lipid Profile

Cord blood, obtained from the umbilical vein and arteries at birth, reflects the metabolic and nutritional status of the fetus. The lipid profile of cord blood includes total cholesterol (TC), a precursor for steroid hormone synthesis and a component of cell membranes; triglycerides (TG), which serve as major energy reservoirs stored in adipocytes; low-density lipoprotein cholesterol (LDL-C), which transports cholesterol to peripheral tissues and is often referred to as “bad cholesterol”; and high-density lipoprotein cholesterol (HDL-C), which facilitates reverse cholesterol transport to the liver and is termed “good cholesterol.” The cord blood lipid profile reflects intrauterine lipid metabolism and placental lipid transfer. Maternal lipids, especially during the third trimester, contribute significantly to fetal lipid levels. However, the placental barrier modulates this transfer, influencing fetal lipid concentrations [74]. Understanding these mechanisms is particularly crucial for SGA neonates, who often experience altered placental function.

Emerging research highlights the role of lipoproteins, apolipoproteins, and lipid precursors in the neonatal lipid profile. These components are not only markers of current metabolic status but also predictors of future metabolic health. The examination of ratios such as LDL-C/HDL-C and non-HDL-C levels offers additional insights into the atherogenic potential of neonatal lipid profiles. Establishing

normative data for these parameters across gestational ages is essential for identifying deviations specific to SGA neonates.[74-75]

Alterations in Lipid Metabolism in SGA Neonates

SGA neonates often exhibit significant deviations in their lipid profile compared to appropriate for gestational age (AGA) counterparts. Studies have frequently reported lower HDL-C levels and higher LDL-C levels in SGA neonates, indicative of an atherogenic lipid profile that may predispose them to long-term cardiovascular risks [6]. These deviations arise primarily from altered metabolic adaptations that occur due to the limited nutrient availability in utero. For instance, the redistribution of blood flow to critical organs, such as the brain, results in reduced fat deposition and lipid storage in peripheral tissues. This phenomenon, known as the "brain-sparing effect," ensures neuroprotection but comes at the cost of impaired peripheral lipid utilization and development.

In SGA neonates, enzymatic activity related to lipid metabolism is notably altered. Lipoprotein lipase (LPL), which plays a pivotal role in triglyceride hydrolysis and lipid storage, is often less active in these neonates. Reduced LPL activity affects the clearance of triglycerides from circulation, resulting in elevated plasma levels of triglycerides and LDL-C. Concurrently, reduced HDL-C levels further exacerbate the lipid profile's atherogenic nature, as HDL-C is essential for reverse cholesterol transport and vascular protection. Hormonal imbalances, such as decreased insulin and leptin levels observed in SGA neonates, further impair lipid metabolism. Insulin acts as a potent regulator of LPL activity and fatty acid synthesis, and its deficiency compromises lipid homeostasis in these neonates.[7]

The epigenetic regulation of genes associated with lipid metabolism is another critical area of alteration in SGA neonates. Intrauterine growth restriction (IUGR) and the associated fetal stress result in epigenetic modifications, such as DNA methylation and histone acetylation, which affect the expression of lipid-metabolism-related genes. Genes involved in fatty acid oxidation, lipid synthesis, and lipoprotein transport are often downregulated, contributing to the dysregulated lipid profile. These epigenetic changes may persist beyond the neonatal period, increasing the susceptibility of SGA individuals to metabolic disorders later in life.

Additionally, altered lipid metabolism in SGA neonates can impact their growth trajectories and energy reserves. Lipids serve as the primary energy source for neonatal growth, and disruptions in lipid utilization can result in poor weight gain and delayed developmental milestones. Addressing these alterations through early nutritional and therapeutic interventions is crucial to mitigate their long-term impacts.

Cord Blood Lipid Profile in AGA vs. SGA Neonates

Several studies have compared lipid profiles in AGA and SGA neonates, highlighting differences that may reflect altered fetal growth and metabolic adaptation:[6]

SGA neonates tend to have **higher TG and lower HDL levels** compared to AGA neonates, suggesting a compensatory response to intrauterine malnutrition.

Studies have reported **lower total cholesterol and LDL levels in SGA neonates**, potentially reflecting reduced hepatic lipid synthesis and altered placental lipid transport.

The altered lipid profile in SGA neonates has been linked to increased risk of metabolic syndrome, insulin resistance, and cardiovascular disease in later life.

Cord Blood Lipid Profile in Term vs. Preterm Neonates

Prematurity is associated with distinct metabolic adaptations that impact lipid profiles:

Preterm neonates often exhibit **lower total cholesterol and LDL levels** compared to term infants, likely due to immature hepatic synthesis.

Higher triglyceride levels have been observed in preterm neonates, possibly due to delayed clearance and immature lipoprotein metabolism.

HDL levels in preterm infants tend to be lower, reflecting immature lipid transport mechanisms and an increased risk of later metabolic disorders.[76]

Influence of Gender on Neonatal Lipid Profiles

Sex-based differences in lipid metabolism have been observed in neonates:

Male neonates often exhibit **higher LDL and triglyceride levels** compared to females.

Female neonates tend to have **higher HDL levels**, which may provide a protective effect against cardiovascular risk in later life.

These differences may be attributed to hormonal influences, differences in fat distribution, and genetic factors.[77]

Maternal and Placental Influences on Cord Blood Lipid Profile

Maternal and placental factors are central to determining the cord blood lipid profile, particularly in SGA neonates. Maternal hyperlipidemia during pregnancy significantly impacts fetal lipid levels. Conditions such as preeclampsia and gestational diabetes mellitus (GDM) alter maternal lipid metabolism, leading to increased circulating triglycerides, cholesterol, and lipoproteins. These maternal lipid levels are partially transferred to the fetus via the placenta, which acts as an active metabolic interface between the mother and fetus [5].

The placenta is a dynamic organ that not only transfers lipids but also actively metabolizes and modifies them. Lipid transfer occurs via specialized transport proteins, such as fatty acid transport proteins (FATPs) and lipoprotein lipase (LPL). In pregnancies complicated by IUGR, which is closely related to SGA, the expression and activity of these proteins are often dysregulated. For example, reduced expression of FATPs in the placenta can limit the uptake and transport of free fatty acids to the fetus, resulting in altered lipid profiles at birth. Similarly, placental LPL activity, crucial for hydrolyzing maternal triglycerides into free fatty acids, is frequently diminished in SGA pregnancies. This dysfunction leads to an inadequate supply of essential fatty acids necessary for fetal growth and development.[44]

The maternal-fetal lipid gradient, a key determinant of fetal lipid availability, is also influenced by maternal diet and metabolic status. In SGA pregnancies, the gradient is often disrupted due to placental insufficiency and reduced perfusion. This disruption compromises the delivery of essential lipids and lipoproteins to the fetus. Moreover, maternal lipoproteins, particularly very-low-density lipoproteins (VLDL) and LDL-C,

exhibit altered interactions with placental receptors, further affecting lipid transfer.[46]

Hormonal regulation plays a pivotal role in modulating placental lipid metabolism. Hormones such as insulin, leptin, and adiponectin, which are influenced by maternal metabolic status, directly impact placental lipid transfer mechanisms. In pregnancies affected by maternal obesity or GDM, increased insulin resistance and altered leptin levels exacerbate placental lipid dysregulation, contributing to the aberrant lipid profiles observed in SGA neonates.

In addition to lipid transport and metabolism, the placenta serves as a barrier to prevent excessive lipid transfer that could harm fetal development. However, in SGA pregnancies, this barrier function is often compromised, leading to suboptimal lipid availability. Understanding the multifaceted role of the placenta in lipid metabolism is critical for devising interventions aimed at improving fetal lipid profiles. Strategies such as optimizing maternal nutrition, improving glycemic control in GDM, and targeting placental transport pathways hold promise for addressing the lipid imbalances in SGA neonates.

Barker's Hypothesis and Its Implications on Neonatal Lipid Profiles

A critical concept in understanding the long-term effects of intrauterine growth on metabolic health is Barker's Hypothesis, also known as the Developmental Origins of Health and Disease (DOHaD) hypothesis. Proposed by Barker and Osmond (1986), this hypothesis suggests that adverse conditions in the intrauterine environment, particularly fetal malnutrition and growth restriction, predispose individuals to chronic diseases later in life, such as cardiovascular disease, hypertension, type 2 diabetes, and metabolic syndrome (Barker, 1995; Hales & Barker, 2001). The hypothesis is particularly relevant in the context of SGA neonates, as these infants experience a suboptimal intrauterine environment that leads to metabolic adaptations designed for survival but may have detrimental effects in adulthood.[1]

According to Barker's Hypothesis, fetal programming occurs in response to intrauterine nutritional deprivation, leading to permanent changes in metabolism, endocrine function, and cardiovascular physiology. This phenomenon is particularly

evident in the lipid metabolism of SGA neonates, who often exhibit higher triglyceride levels, lower HDL levels, and altered cholesterol transport mechanisms (Singhal et al., 2003; Kensara et al., 2006).[77-78] These adaptations, while beneficial for survival in a nutrient-limited intrauterine environment, increase the risk of dyslipidemia, insulin resistance, and cardiovascular complications in adulthood (Barker et al., 2002).[1] Additionally, preterm infants, who face metabolic immaturity at birth, may undergo similar long-term consequences due to altered lipid processing and energy metabolism in early life.[80]

Research has demonstrated that SGA neonates who experience rapid postnatal weight gain—a phenomenon known as catch-up growth—are at an even greater risk of developing metabolic disorders.[81] This rapid compensatory growth leads to excessive fat accumulation, increased insulin resistance, and an unfavorable lipid profile, which aligns with the observations made in adults who were born SGA. Furthermore, studies have indicated that low birth weight, in conjunction with adult obesity, exponentially increases the risk of metabolic syndrome, reinforcing the predictive value of neonatal lipid profiles in long-term health outcomes .[1]

In the context of term vs. preterm neonates, Barker's Hypothesis further explains how early-life metabolic programming impacts long-term health trajectories. Preterm infants, due to their immature hepatic function and disrupted placental nutrient supply, often exhibit lower cholesterol levels and impaired lipid transport, which may influence lipid metabolism throughout life. Additionally, the mismatch between prenatal malnutrition and postnatal overnutrition (often seen in preterm and SGA infants receiving nutritional support) can further amplify the risk of metabolic derangements.[80]

The integration of Barker's Hypothesis into neonatal lipid research provides a critical perspective on the lifelong implications of intrauterine growth patterns. By identifying lipid profile alterations in SGA and preterm infants, clinicians can take proactive steps to implement early nutritional interventions, lifestyle modifications, and metabolic monitoring to mitigate long-term health risks. Future studies should continue to explore the epigenetic mechanisms underlying fetal programming, as well as develop targeted strategies to optimize postnatal growth while minimizing the risk of

metabolic syndrome in at-risk neonates.[1]

Lipid Profile in Neonates: Components and Normal Ranges

Lipid metabolism in neonates differs significantly from that in adults due to **placental lipid transport**, **hepatic immaturity**, and **unique metabolic demands**. The normal lipid profile values in neonates, based on available literature, are as follows:[1]

Lipid Parameter	**Normal Range in Neonates (mg/dL)**
Total Cholesterol (TC)	70 – 170
Triglycerides (TG)	30 – 120
High-Density Lipoprotein (HDL)	25 – 80
Low-Density Lipoprotein (LDL)	30 – 130

Neonatal lipid levels **vary significantly based on gestational age, intrauterine growth status, and gender**, as discussed above .

Neonatal Blood Cholesterol Screening (NCEP Expert Panel Guidelines)

The National Cholesterol Education Program (NCEP) Expert Panel provides guidelines for cholesterol screening in neonates and children, emphasizing early detection of dyslipidemia.

Recommendations for Neonates and Children

- Universal cholesterol screening is not routinely performed in neonates.
- Screening is recommended in neonates with a family history of hyperlipidemia, early cardiovascular disease, or metabolic syndrome.
- Lipid testing is advised in infants with SGA, preterm birth, or metabolic disorders.

NCEP Cholesterol Risk Categories for Children (≥2 Years Old)[82]

Lipid Parameter	Acceptable (mg/dL)	Borderline (mg/dL)	High (mg/dL)
Total Cholesterol	<170	170 – 199	≥200
LDL	<110	110 – 129	≥130
Triglycerides (0-9 years)	<75	75 – 99	≥100
Triglycerides (10-19 years)	<90	90 – 129	≥130
HDL	>45	40 – 45	<40

For neonates at risk, early lipid screening may be considered, especially in cases of SGA, preterm birth, or a strong family history of dyslipidemia.

Treatment of Hyperlipidemia in Neonates and Children

Neonatal hyperlipidemia is uncommon but can occur due to genetic conditions (familial hypercholesterolemia), prematurity, or parenteral nutrition.[82]

Treatment Strategies

- Dietary modifications: Breastfeeding is recommended as human milk provides essential long-chain polyunsaturated fatty acids (LCPUFAs) that aid in brain development and lipid metabolism.
- Formula adjustments: Special lipid formulations may be required for preterm infants or those on parenteral nutrition.
- Pharmacologic interventions: Rarely used in neonates but may be considered in older children with severe familial hypercholesterolemia.

Long-Term Follow-Up

Infants with **persistent dyslipidemia** should undergo **regular lipid monitoring** and receive early lifestyle interventions to prevent long-term cardiovascular risks.[6]

Clinical Implications of Cord Blood Lipid Profiles in SGA

The lipid profile in cord blood serves as a biomarker for assessing fetal metabolic health. Key clinical implications include predicting neonatal outcomes, as abnormal lipid levels are associated with adverse outcomes such as hypoglycemia, hypothermia, and feeding difficulties in SGA neonates; assessing long-term risks, as dyslipidemia in SGA neonates can indicate future risks for obesity, type 2 diabetes, and cardiovascular diseases; and guiding nutritional interventions, as understanding lipid metabolism can help optimize postnatal nutrition to prevent metabolic complications.

Recent studies:

Ramanathan et al. (2024) conducted a study to facilitate the early detection of lipid profile abnormalities at birth, focusing particularly on preterm and small for gestational age (SGA) neonates. The research spanned three years, from 2020 to 2023, and included 222 neonates in the neonatal intensive care unit (NICU) of the Department of Pediatrics at Government Medical College, Chidambaram. The cohort consisted of 155 term neonates, 67 preterm neonates, and six post-term neonates. Males accounted for the majority (51.5%), although there was a higher proportion of female term neonates (60.8%) and male preterm neonates (44.7%). Among appropriate for gestational age (AGA) neonates, males were predominant (60.2%), whereas females were more common among SGA neonates (48.5%). However, no statistically significant gender differences were observed between groups. Birth weight was significantly lower in SGA neonates (1.81±0.34 kg) compared to AGA neonates (2.82±0.25 kg) ($p<0.05$), although other neonatal variables such as length and head circumference did not differ significantly between the two groups.\n\nPreterm neonates exhibited higher cord lipid profile values than their term counterparts. Similarly, SGA neonates had elevated cord lipid profile parameters compared to AGA neonates, except for HDL levels (32.77±09.57), which were lower in the SGA group. The ponderal index demonstrated a significant correlation with

cord blood lipid profiles ($p<0.05$). Asymmetrical intrauterine growth restriction (IUGR) neonates had elevated triglyceride (TG) levels (86.70±17.72) and very low-density lipoprotein (VLDL) levels (17.34±3.54). In preterm neonates, the cord lipid profile values were TG (80.75±12.84), total cholesterol (TC) (98.91±26.60), HDL (34.56±05.35), LDL (53.77±25.25), and VLDL (16.15±04.30). Term neonates showed lower values: TC (82.63±27.16), TG (71.21±22.35), HDL (28.36±06.29), LDL (39.85±14.10), and VLDL (14.43±04.47), with statistically significant differences between groups ($P<0.01$).\n\nFor AGA neonates, the cord lipid profile values were TC (86.08±25.49), TG (82.96±19.82), HDL (28.98±07.76), LDL (40.75±15.66), and VLDL (16.15±04.30). In contrast, SGA neonates exhibited TC (83.99±28.64), TG (70.32±22.33), HDL (32.77±09.57), LDL (48.57±22.88), and VLDL (14.43±04.47). These differences were statistically significant ($p<0.05$), except for HDL, which was lower in SGA neonates. Asymmetrical IUGR neonates displayed the following cord lipid profile values: TC (93.55±22.45), TG (86.70±17.72), LDL (47.23±17.49), HDL (28.89±7.51), and VLDL (17.34±3.54). Symmetrical IUGR neonates had slightly different profiles: TC (103.92±32.65), TG (71.38±21.86), LDL (45.28±7.72), HDL (29.28±8.68), and VLDL (14.39±4.39). [83]

A another study, Mantry et al. (2024) compared the cord blood lipid profiles of 50 SGA neonates and 50 AGA neonates in a cross-sectional study. Information was gathered through detailed histories, clinical examinations, and pre-designed questionnaires. Lipid profiles for both groups were analyzed and compared. The study revealed no significant differences in lipid profiles based on gender. However, SGA neonates exhibited significantly higher levels of serum total cholesterol (TC), LDL, and TG compared to AGA neonates. Among low birth weight (LBW) babies, TC levels were notably higher compared to normal birth weight (NBW) babies. HDL levels, however, did not show any significant differences between SGA and AGA neonates.[84]

Machakanur et al. (2022) conducted a comparative study on lipid profiles of small for gestational age (SGA) and appropriate for gestational age (AGA) neonates. This case-control study took place in the Department of Pediatrics, Obstetrics, and Gynecology at Karwar Institute of Medical Sciences, Karnataka, India. A total of 133 deliveries were randomly included, with 99 AGA and 34 SGA neonates. The findings indicated

that SGA neonates had a significantly lower mean gestational age (37.69±2.45 weeks) compared to AGA neonates (38.55±1.11 weeks; p=0.022). The mean total cholesterol (TC) was higher in SGA neonates (63.62±40.48 mg/dL) compared to AGA neonates (48.69±2.29 mg/dL), a difference that was statistically significant (p=0.007). While HDL levels were comparable between the groups (21.82±13.26 mg/dL in SGA and 21.49±14.64 mg/dL in AGA), the mean VLDL levels were significantly higher in SGA neonates (17.11±25.35 mg/dL) than in AGA neonates (9.47±9.35 mg/dL; p=0.012).[85]

Amandeep et al. (2019) investigated cord blood lipid levels in neonates with varying gestational ages (GA), weights, and sexes. The study included 1,000 healthy term and preterm neonates after parental consent. The New Ballard Score confirmed GA, and Fenton's growth charts classified neonates as SGA, AGA, or large for gestational age (LGA). Lipid profiles were assessed using an enzymatic colorimetric method, and LDL cholesterol was calculated via Friedewald's formula. The study revealed that preterm neonates (GA 28–36 weeks) had higher levels of TC, LDL, VLDL, and triglycerides compared to term neonates, while HDL levels were significantly lower in preterm neonates. A decline in lipid fractions was observed as birth weight increased from <1.5 kg to ≥2.5 kg. Female neonates exhibited higher lipid levels compared to males, with HDL differences reaching statistical significance (p<0.001).[86]

Arora et al. (2017) conducted a study to compare cord blood lipid profiles between term SGA and AGA neonates, emphasizing the importance of early lipid profile screening for assessing cardiovascular risk. Out of 200 full-term neonates studied at SGRDIMSR, 100 were AGA and 100 were SGA. The mean lipid profile values in AGA neonates were TC (72.29±21.13 mg/dL), triglycerides (67.18±20.36 mg/dL), HDL (28.51±5.68 mg/dL), LDL (34.94±16.45 mg/dL), and VLDL (13.43±4.10 mg/dL). SGA neonates demonstrated higher values for TC (100.85±26.82 mg/dL), triglycerides (82±22.58 mg/dL), LDL (44.93±7.66 mg/dL), and VLDL (16.40±4.48 mg/dL), but HDL was slightly lower (27.70±7.79 mg/dL).[87]

Lobo et al. (2016) explored the impact of in utero malnutrition on cardiovascular risks by comparing cord blood lipid profiles and insulin levels in SGA and AGA neonates.

This study involved full-term neonates born at an Obstetric Unit in Secunderabad, India. The SGA group (n=51) showed significantly higher levels of cholesterol, triglycerides, and LDL compared to the AGA group (n=52; $p<0.01$). However, no significant differences were found in HDL or insulin levels between the groups. Notably, severe maternal anemia was observed only in the SGA group, while mild to moderate anemia was present in mothers from both groups.[88]

MATERIALS AND METHODS

MATERIAL AND METHODS

Study design: Cross-sectional study

Study Place: Conducted in NICU Department of Pediatrics of Santosh hospital

Minimum Sample Size Required: 146 per group , (with 95% confidence interval and 5% margin of error)

Sampling Frame: 146 per group , Total-292

Used Formula

n1 = (s12 +s2 /k)(z1-a /2 + z1-b)2 /D2

n2 =(k*s12 +s2)(z1-a/2 +z1-b)2 /D2

The notation for the formulae are: n1 = sample size of Group 1
n2 = sample size of Group 2
s1 = standard deviation of Group 1 s 2 = standard deviation of Group 2

D = difference in group means

k = ratio = n2/n1
Z1-α/2 = two-sided Z value (eg. Z=1.96 for 95% confidence interval). Z1-β = power

Ethical Clearance: Ethical clearance was taken from the ethical committee at Santosh Medical College and Hospital, Ghaziabad.

Inclusion Criteria:

Term neonates

-Appropriate for gestational age

-Small for gestational age

2) Preterm neonates

-Appropriate for gestational age

-Small for gestational age

3) Ponderal index

-*<2 in between 29-37 weeks*

-<2.25 in more than 37 weeks

Exclusion Criteria:

- Neonates with congenital malformations
- Neonates born to mother with maternal illness like diabetes , tb , asthma , pregnancy induced hypertension, family history of coronary artery diseases
- Any maternal medication except vitamins and iron supplements
- Neonates with birth asphyxia , APGAR score <7
- Large for gestational age babies >90th percentile
- Post term neonates >42 weeks \

METHOD OF DATA COLLECTION

A cross-sectional study was conducted in the NICU of Santosh Hospital on neonates born via normal vaginal delivery and caesarean section.

Informed written consent was obtained from the parents or guardians of the neonates. A 5 ml sample of cord blood was collected from the umbilical cord immediately after delivery, specifically from the placental end.

The following anthropometric measurements were performed on the same participants:

- **Length:** An infantometer was used to measure the length of the neonate. The neonate was placed on the infantometer with legs fully extended, and the movable foot end was adjusted to the soles of the feet to record the length.
- **Weight:** The weight of the neonate was recorded using an electronic weighing machine, without any clothing, immediately after birth.
- **Head circumference:** Head circumference was measured using a non-elastic measuring tape by determining the greatest occipitofrontal circumference.

- **Ponderal Index (PI):** PI was calculated using the formula:
  PI = 100 × Weight (grams) / Length $(cm)^3$.

It was used to define intrauterine growth retardation, with the following cut-offs:

- PI < 2 for neonates between 29–37 weeks of gestation
- PI < 2.25 for neonates beyond 37 weeks of gestation.

STATISTICAL ANALYSIS:

The data were initially checked for completeness, and data was cleaned for errors and missing values. The corrected data was then entered into Microsoft Excel after preparing a Master-chart. After data entry of every ten questionnaires, one random form was picked and data entry was re-checked. An independent person verified data entry of two randomly chosen forms after entry of every fifth questionnaire.

Data analysis was done using licensed SPSS software version 24.0 (Chicago, Illinois). Univariate analyses was done initially and the results were presented with the help of tables,. Descriptive statistics were used to calculate frequencies of categorical variables, and measures of central tendencies and dispersion were used to describe continuous variables. Independent t-test was used to compare the continuous variable and chi-square test was used for categorical variables. A p-value <0.05 was considered as statistically significant.

ETHICAL ISSUES:

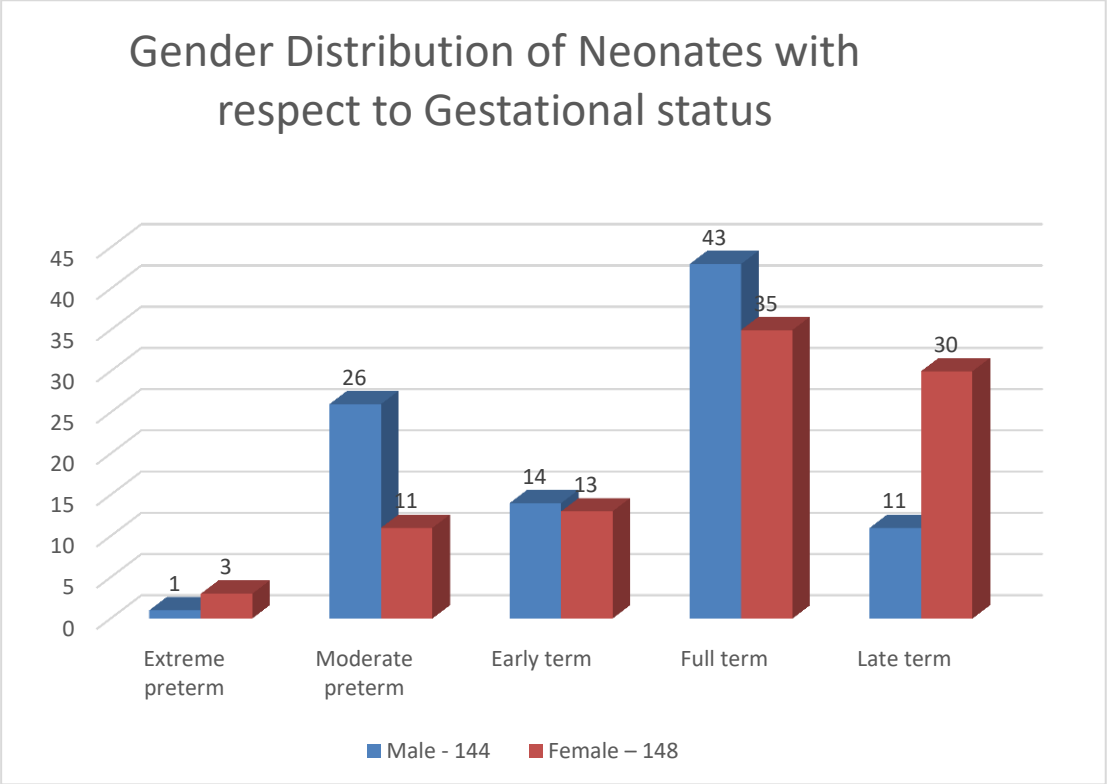
Approval from Institutional Ethical Committee of the hospital was taken before the start of the study. Written and informed consent taken from the participants before proceeding the study. Each eligible subject was explained about the purpose of the study by the investigator and an informed consent was obtained, prior to inclusion. They were assured of complete confidentiality of information, and the option of withdrawing from the study at any point of time. The study did not involve any method that puts the subjects, family members or the investigator at risk.

OBSERVATIONS AND RESULTS

OBSERVATIONS AND RESULTS

Table no 1 Gender Distribution of Neonates with respect to Gestational status

Gestational age	**Number of Males**		**Number of Females**		**Total**	
	n= 144	**%=49.3%**	**n= 148**	**%=50.6%**	**n= 292**	**%=100%**
Extreme preterm	1	0.6%	3	2.0%	4	1.3%
Very preterm	3	2.0%	7	4.8%	10	3.4%
Moderate preterm	26	18.0%	11	7.6%	37	12.6%
Late preterm	46	31.9%	49	33.1%	95	32.5%
Early term	14	9.7%	13	8.7%	27	9.2%
Full term	43	29.8%	35	23.6%	78	26.7%
Late term	11	7.6%	30	20.2%	41	14.0



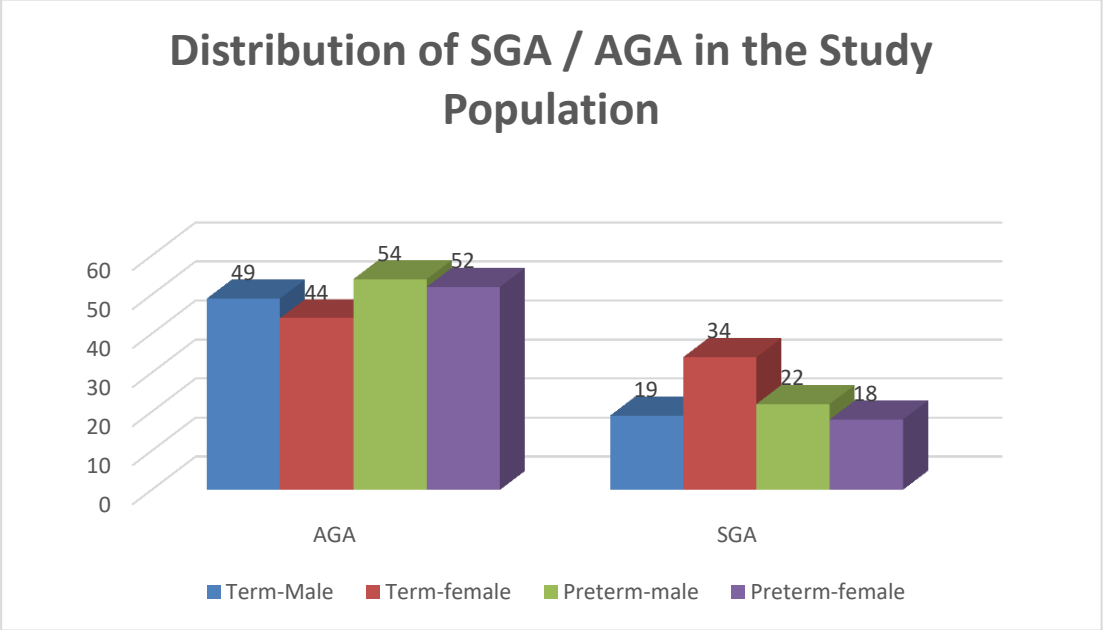
Among 292 neonates the distribution of male that is 49.3 % and female is 50.6% were nearly equal as shown in table no 1. The largest group was late preterm (95; 32.5%), followed by full term (78; 26.7%) and late term (41; 14%). The mean value and standard deviation for gestational age of term neonates was 38.71 ± 1.20 weeks and the mean value and standard deviation for gestational age of preterm neonates was 34 ± 2.05 weeks.

Table 2 . Distribution of SGA / AGA in the Study Population

SGA/AGA	Term n=146			Preterm n=146		
	Male	**Females**	**Total**	**Male**	**Female**	**Total**
AGA*	49 (33.5%)	44 (30.1%)	93 (63.6%)	54 (36.9%)	52 (35.6%)	106 (72.6%)
SGA**	19 (13.0%)	34 (23.2%)	53 (36.3%)	22 (15.0%)	18 (12.3%)	40 (27.3%)
Total	68 (46.5%)	78 (53.4%)	146 (100%)	76 (52.0%)	70 (47.9%)	146 (100%)

***AGA- Appropriate for Gestational Age**

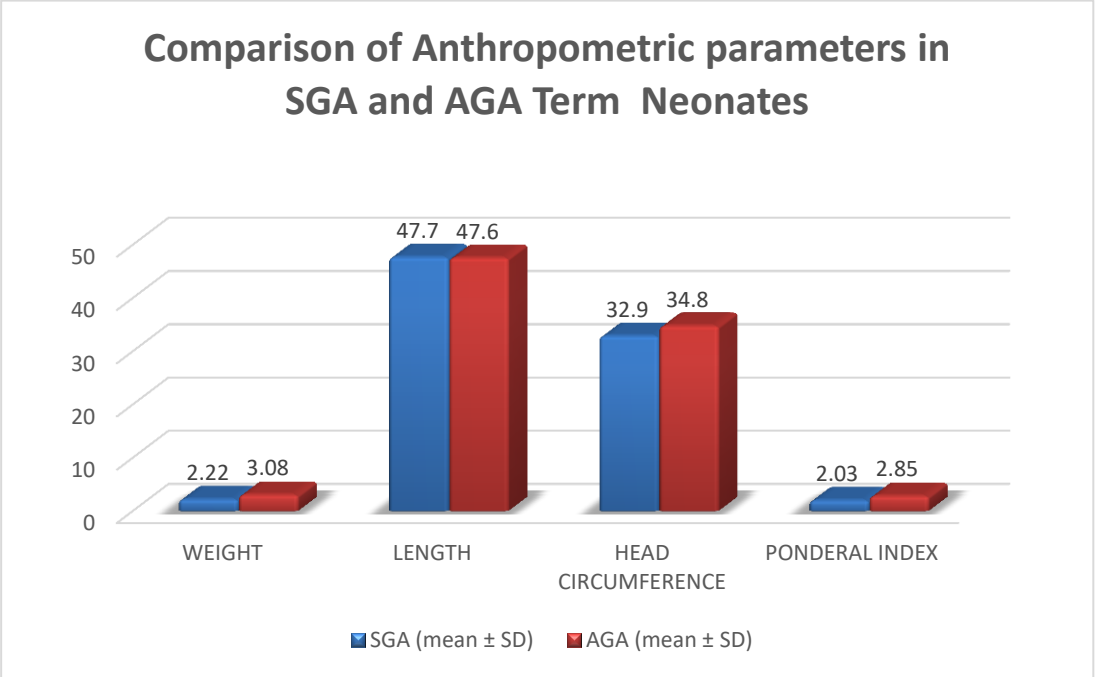
****SGA- Small for Gestational Age**



In term neonates, 93 (63.6%) were AGA and 53 (36.3%) were SGA. In preterm neonates, 106 (72.6%) were AGA and 40 (27.3%) were SGA. The distribution of males and females in both that is Term AGA neonates and Preterm AGA neonates showed no significant difference that is males accounting for 33.5% male Term AGA neonates and 30.1% female Term AGA neonates followed by *36.9%* male preterm AGA neonates and 35.6% female preterm AGA neonates . The distribution of AGA was more common in both term (63.6%)and preterm (72.6%) groups as shown in table no 2

Table 3 (a) Comparison of Anthropometric parameters in SGA and AGA Term Neonates

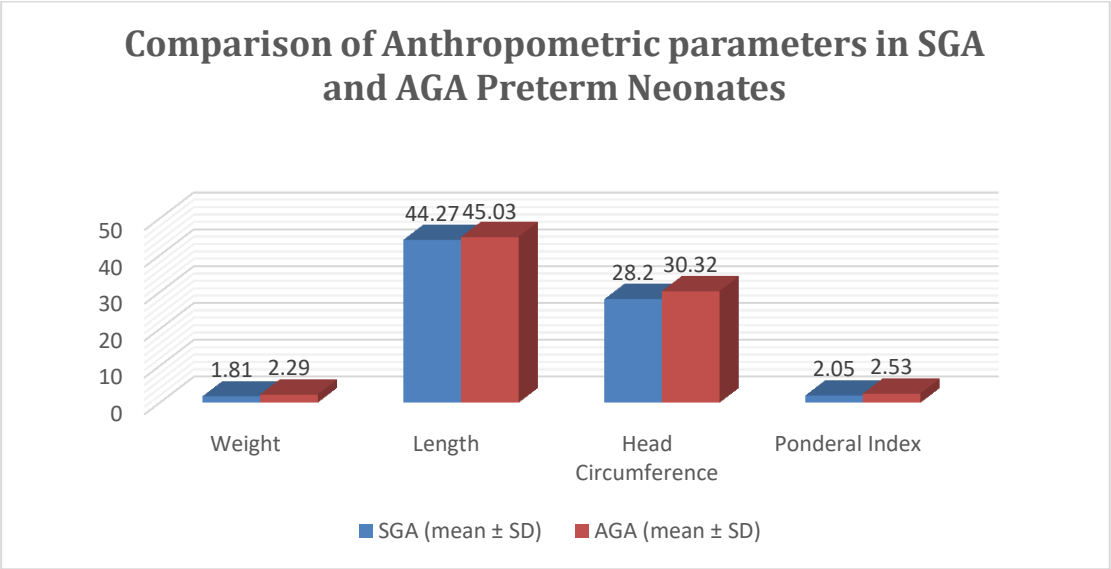
Anthropometric parameters	**SGA (mean ± SD)**	**AGA (mean ± SD)**	**p-VALUE**
WEIGHT	2.22 ± 0.28 kgs	3.08 ± 0.35 kgs	0.0001
LENGTH	47.7 ± 2.12 cms	47.6 ± 1.86 cms	0.569
HEAD CIRCUMFRENCE	32.9 ± 1.34 cms	34.8 ± 1.27 cms	0.034
PONDERAL INDEX	2.03 ± 0.21	2.85 ± 0.31	0.0123



The mean value and standard deviation [SD] of birth weight in term SGA neonates was 2.22 ± 0.28 and the mean value and standard deviation [SD] of birth weight in term AGA was 3.08 ± 0.35 which showed a significant difference(p value-0.0001) The mean head circumference [SD] in term SGA was 32.9 ± 1.34 and term AGA was 34.8 ± 1.27 which also showed significant difference (p value 0.034) . Mean value [SD] of ponderal index distribution as shown in table 3 (b) for term SGA 2.03 ± 0.21 in comparison to term AGA 2.85 ± 0.31 was significant p value - 0.0123). The mean length [SD] one parameter in term AGA vs SGA in anthropometry which did not show any significant difference (p=0.569**).**

Table 3 (b) Comparison of Anthropometric parameters in SGA and AGA Preterm Neonates

Anthropometric parameters	SGA (mean ± SD)	AGA (mean ± SD)	p-VALUE
WEIGHT	1.81 ± 0.45 kgs	2.29 ± 0.49 kgs	0.0001
LENGTH	44.27 ± 4.27 cms	45.03 ± 3.34 cms	0.582
HEAD CIRCUMFRENCE	28.20 ± 2.43 cms	30.32 ± 1.90 cms	0.0342
PONDERAL INDEX	2.05 ± 0.26	2.53 ± 0.48	0.012

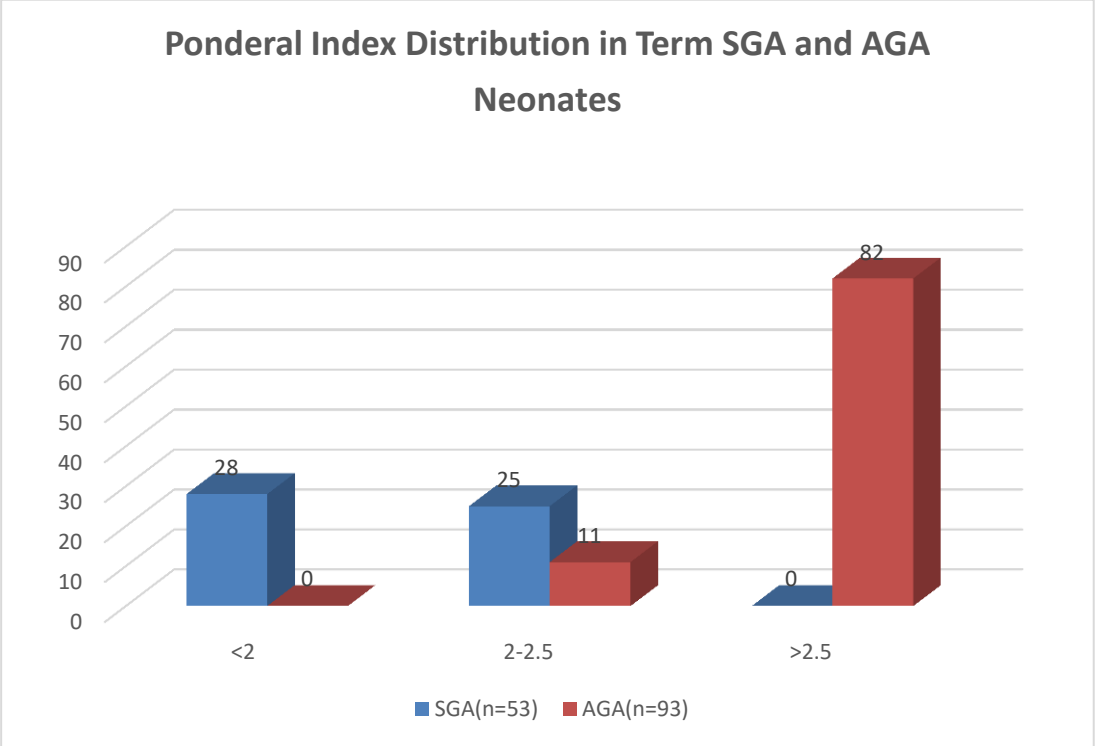


***Weight in grams **Length in centimetre *** head circumference in centimetre**

The mean value and standard deviation [SD] of birth weight in Preterm SGA neonates was 1.81 ± 0.45 and the mean value and standard deviation [SD] of birth weight in Preterm AGA was 2.29 ± 0.49 which showed a significant difference(p value-0.0001) The mean head circumference [SD] in preterm SGA was 28.20 ± 2.43 and preterm AGA was 30.32 ± 1.90 which also showed significant difference (p value 0.0342) . Mean value [SD] of ponderal index distribution as shown in table 3 (b) for preterm SGA 2.05 ± 0.26 in comparison to preterm AGA 2.53 ± 0.48 was significant p=0.012). The mean length [SD] one parameter in anthropometry which did not show any significant difference (p=0.582).

Table 4(a) Ponderal Index Distribution in Term SGA and AGA Neonates

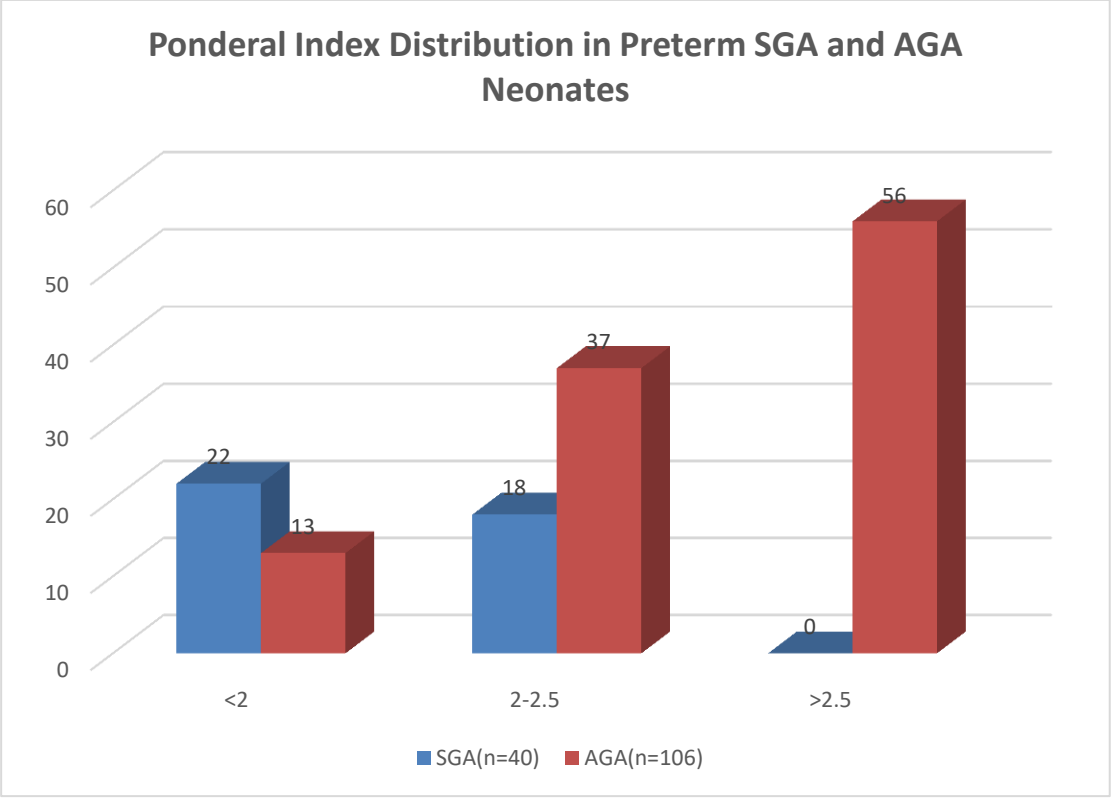
Ponderal index	SGA(n=53)		AGA(n=93)	
	n	%	n	%
<2	28	52.8%	0	0.0%
2-2.5	25	47.1%	11	11.8%
>2.5	0	0.0%	82	88.1%
Total	53	100%	93	63.6%



Among SGA term neonates, 52.8% had a ponderal index <2, and 47.1% had 2–2.5. In contrast, 88.1% of AGA neonates had a ponderal index >2.5. No SGA neonates had index >2.5 as it is usually seen in only AGA or Term neonates

Table 4(b) Ponderal Index Distribution in Preterm SGA and AGA Neonates

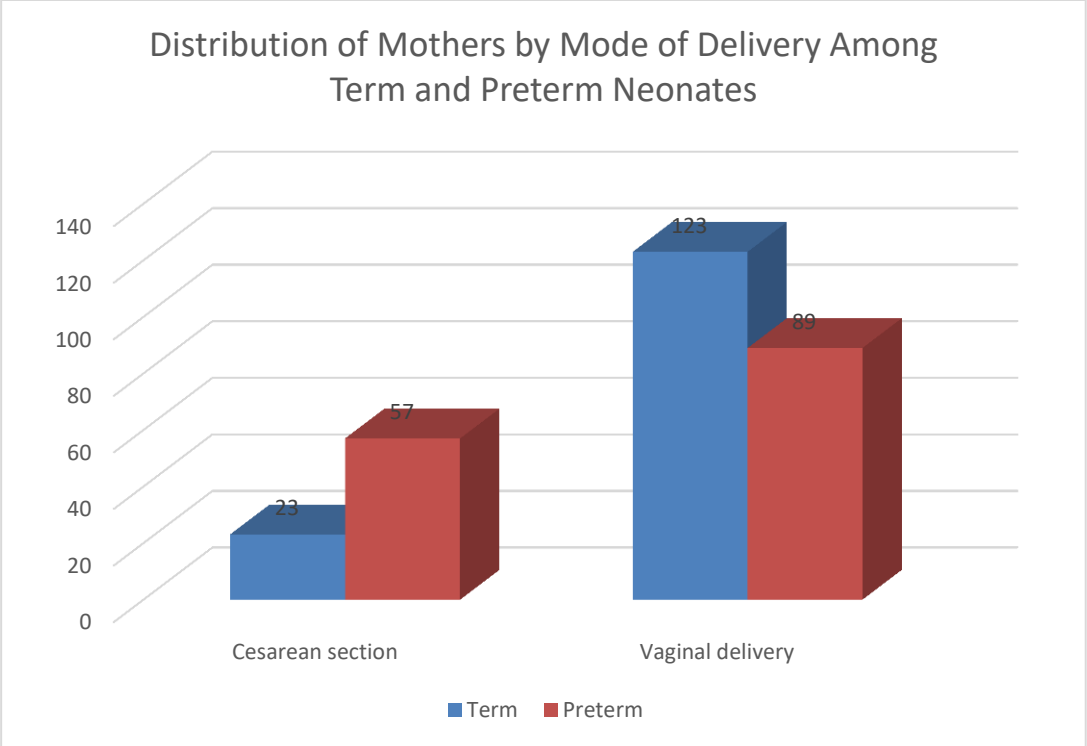
Ponderal index	SGA(n=40)		AGA(n=106)	
	n	%	n	%
<2	22	55%	13	12.2%
2-2.5	18	45%	37	34.9%
>2.5	0	0.0%	56	52.8%
Total	40	100%	106	100%



In the preterm group, 55% of SGA neonates had a ponderal index <2, and none had >2.5. Among AGA neonates, 52.8% had a ponderal index >2.5, highlighting clear nutritional disparities.

Table 5 Distribution of Mothers by Mode of Delivery Among Term and Preterm Neonates

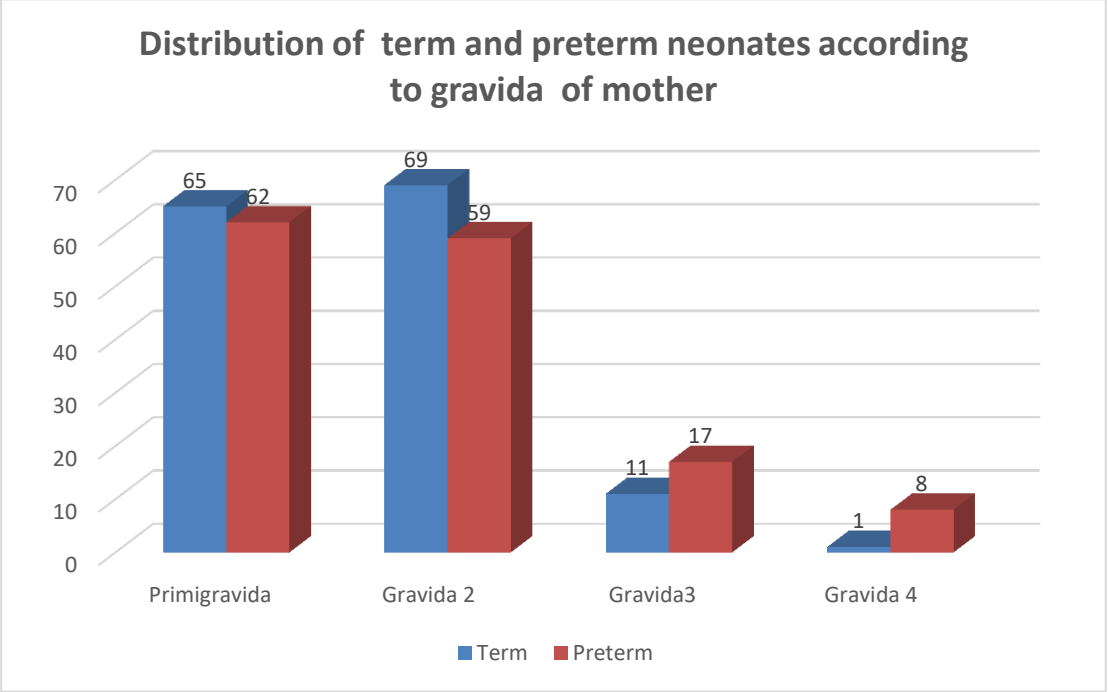
Mode of delivery	Term		Preterm		p-value
	Count	%	Count	%	
Cesarean section	23	15.8%	57	39.0%	0.00001
Vaginal delivery	123	84.2%	89	61.0%	
Total	146	100.0%	146	100.0%	



Cesarean section was more frequent in preterm neonates (39%) compared to term neonates (15.8%), and this difference was statistically significant (p=0.00001).

Table 6: Distribution of term and preterm neonates according to Gravida of mother

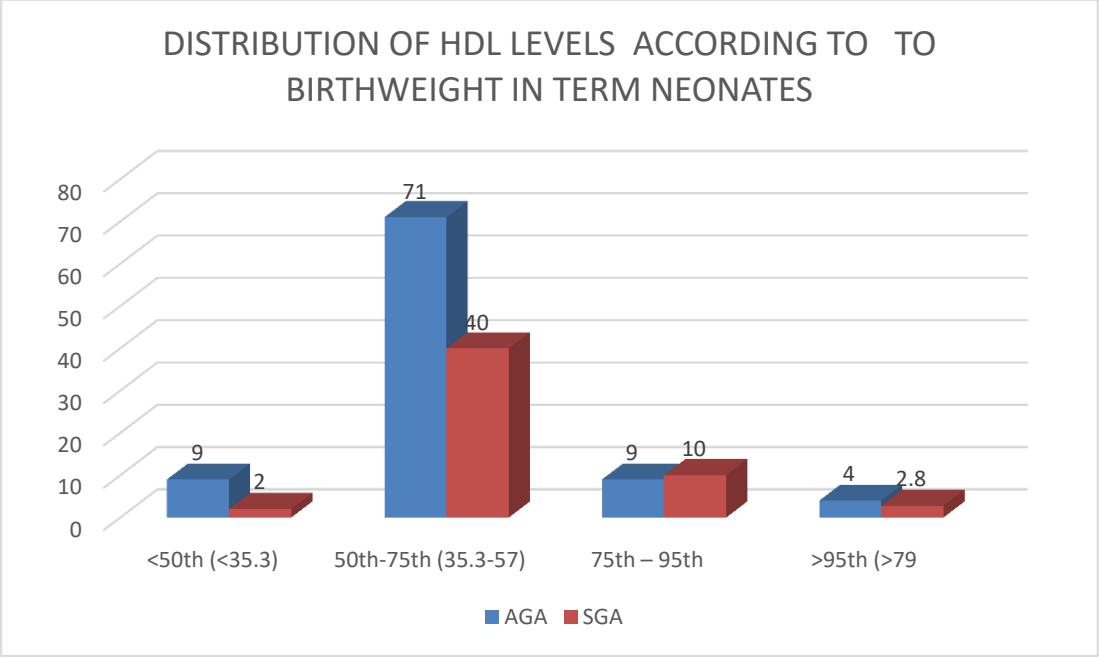
Gravida	Term		Preterm		p-value
	Number	%	Number	%	
Primi	65	44.5%	62	42.4%	0.055
Multigravida	n= 81	55.4%	n = 84	57.5%	
G2	69	47.2%	59	40.4%	
G3	11	7.5%	17	11.6%	
G4	1	0.6%	8	5.4%	
Total	146	100.0%	146	100.0%	



Percentage of mothers that delivered term Neonates were 55.4% in comparison to the multigravida mothers delivering preterm neonates that was 57.4% . Hence and the distribution of gravida showed no significant difference (p=0.055).

Table 7 (a) PERCENTILE DISTRIBUTION OF HDL LEVELS AND THEIR COMAPRISON IN TERM AGA AND SGA NEONATES

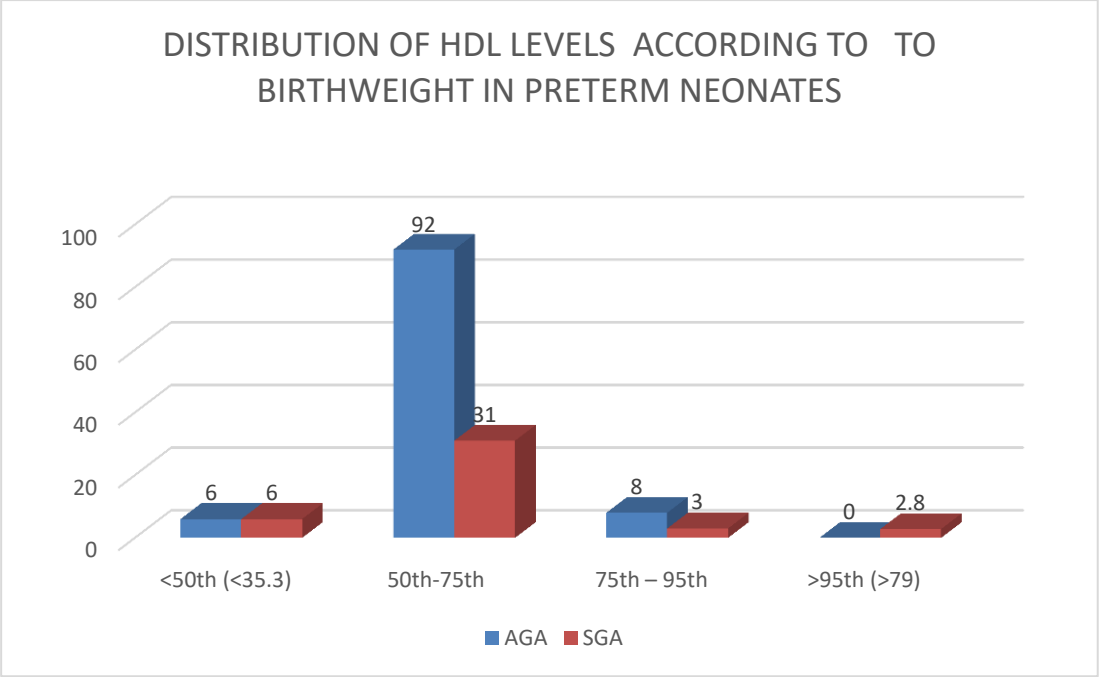
DISTRIBUTION OF HDL LEVELS ACCORDING TO TO BIRTHWEIGHT IN TERM NEONATES						
	VALUES (mg/dl)		GROUP		TOTAL	P-VALUE
			AGA	SGA		
HDL LEVELS	<50th (<35.3)	n	9	2	11	0.0001
		%	9.6%	3.7%	7.5%	
	50th-75th (35.3-57)	n	71	40	111	
		%	76.3%	75.4%	76.0%	
	75th – 95th (58-79)	n	9	10	19	
		%	9.6%	18.8%	13.0%	
	>95th (>79)	n	4	1	5	
		%	4.3%	1.8%	3.4%	
Total		n	93	53	146	
		%	100%	100%	100%	



Level of HDL In term SGA neonates was between 75th–95th percentile (18.8%) as compared to the tem AGA neonates accounting for 9.6% . Very few were below the 50th percentile. The difference was significant (p=0.0001).

Table 7 (b) PERCENTILE DISTRIBUTION OF HDL LEVELS AND THEIR COMAPRISON IN PRETERM AGA AND SGA NEONATES

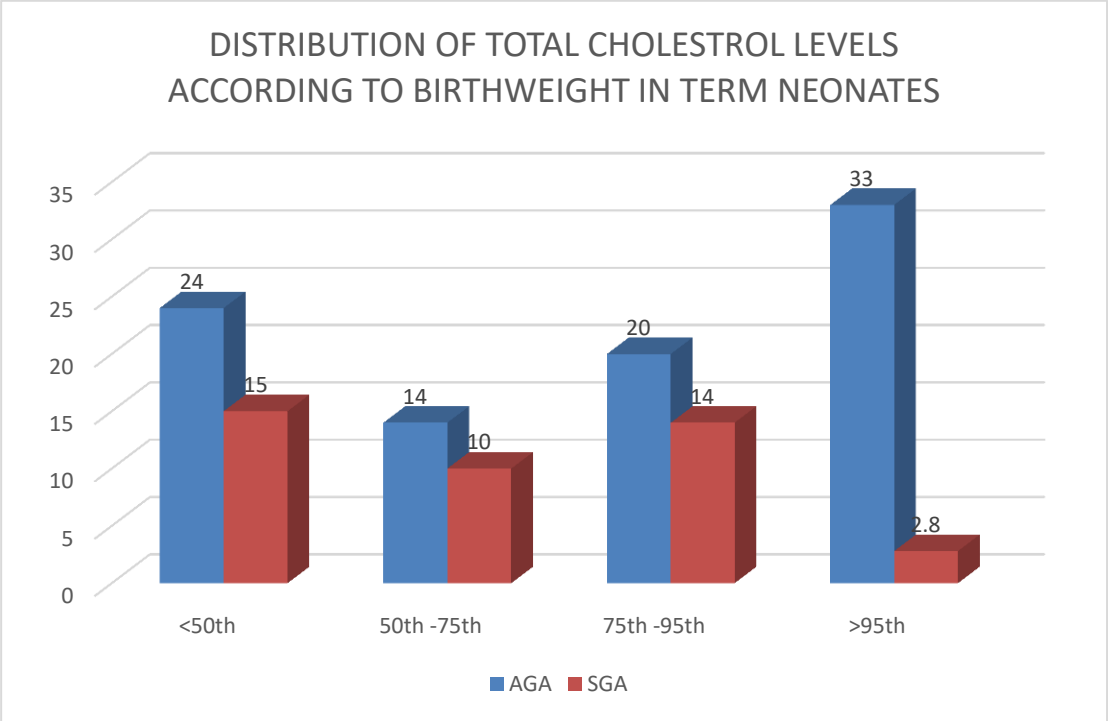
DISTRIBUTION OF HDL LEVELS ACCORDING TO PRETERM AGA AND SGA NEONATES						
	VALUES (mg/dl)		GROUP		TOTAL	P-VALUE
			AGA	SGA		
HDL LEVELS	<50th (<35.3)	n	6	6	12	0.0001
		%	5.6%	15%	8.2%	
	50th-75th (35.3-57)	n	92	31	123	
		%	86.7%	77.5%	84.2%	
	75th – 95th (58-79)	n	8	3	11	
		%	7.5%	7.5%	7.5%	
	>95th (>79)	n	0	0	0	
		%	0%	0%	0%	
Total		n	106	40	146	
		%	100%	100%	100%	



Among preterm neonates, 15% of SGA neonates were below the 50th percentile for HDL compared to 5.6% of AGA. No neonate was above the 95th percentile. The distribution was statistically significant (p=0.0001).

Table 8 (a) PERCENTILE DISTRIBUTION OF TOTAL CHOLESTROL LEVELS AND THEIR COMAPRISON IN TERM AGA AND SGA NEONATES

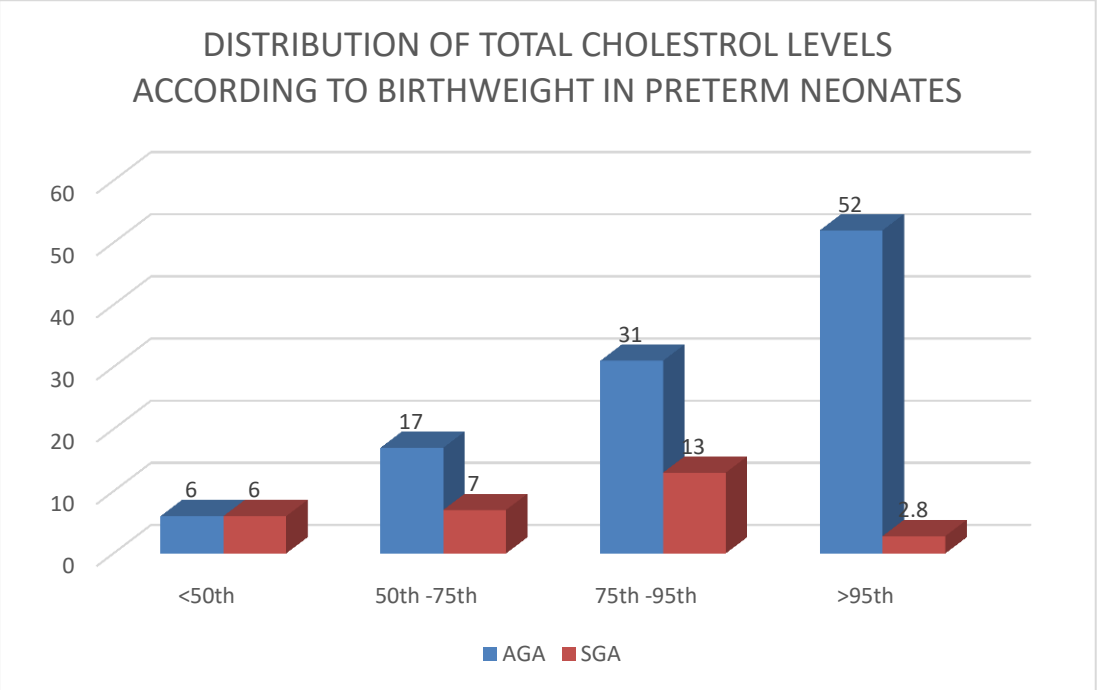
DISTRIBUTION OF TOTAL CHOLESTROL LEVELS ACCORDING TO BIRTHWEIGHT IN TERM NEONATES						
	VALUES (mg/dl)		**GROUP**		**TOTAL**	**P-VALUE**
			AGA	**SGA**		
Total Cholesterol Levels	<50th (<150)	n	24	15	39	**0.0021**
		%	25.8%	28.3%	26.7%	
	50^{th} -75^{th} (150-200)	n	14	10	24	
		%	15%	18.8%	16.4%	
	75^{th} -95^{th} (201-249)	n	20	14	34	
		%	21.9%	26.4%	23.2%	
	>95^{th} (>250)	n	33	14	47	
		%	36.3%	26.4%	32.1%	
Total		n	91	53	146	
		%	100%	100%	100%	



AGA neonates showed higher levels Of total cholesterol that is in the >95th percentile (36.3%) in comparison to SGA (26.4%). The distribution showed significant differences (p=0.0021).

Table 8 (b) PERCENTILE DISTRIBUTION OF TOTAL CHOLESTROL LEVELS AND THEIR COMAPRISON IN PRETERM AGA AND SGA NEONATES

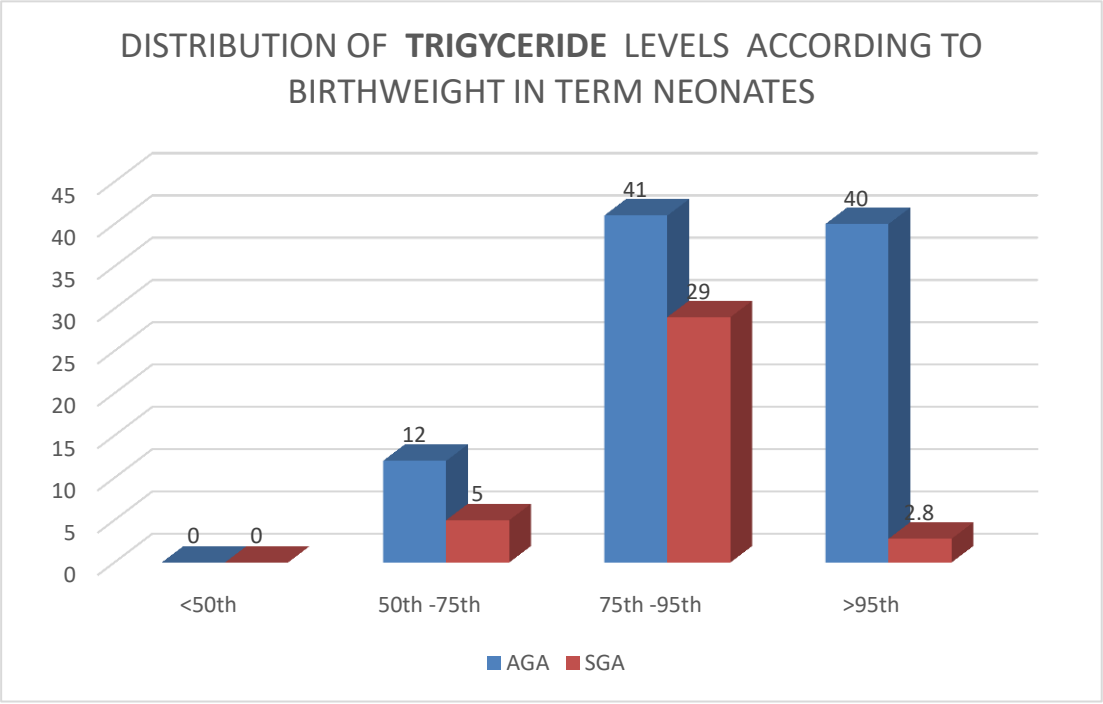
DISTRIBUTION OF TOTAL CHOLESTROL LEVELS ACCORDING TO PRETERM AGA AND SGA NEONATES						
	VALUES (mg/dl)		GROUP		TOTAL	P-VALUE
			AGA	SGA		
Total cholesterol Levels	<50th (<150)	n	6	6	12	0.0001
		%	5.6%	15%	8.2%	
	50^{th} -75^{th} (150-200)	n	17	7	24	
		%	16.0%	17.5%	16.4%	
	75^{th} -95^{th} (201-249)	n	31	13	44	
		%	29.2%	32.5%	30.1%	
	>95^{th} (>250)	n	52	14	66	
		%	49.0%	35.0%	45.2%	
Total		n	106	40	146	
		%	100%	100%	100%	



A greater proportion of AGA neonates had total cholesterol >250 mg/dL (49%) compared to SGA (35%). This difference was statistically significant (p=0.0001).

Table 9 (a) PERCENTILE DISTRIBUTION OF TRIGYCERIDES VALUES AND THEIR COMAPRISON IN TERM AGA AND SGA NEONATES

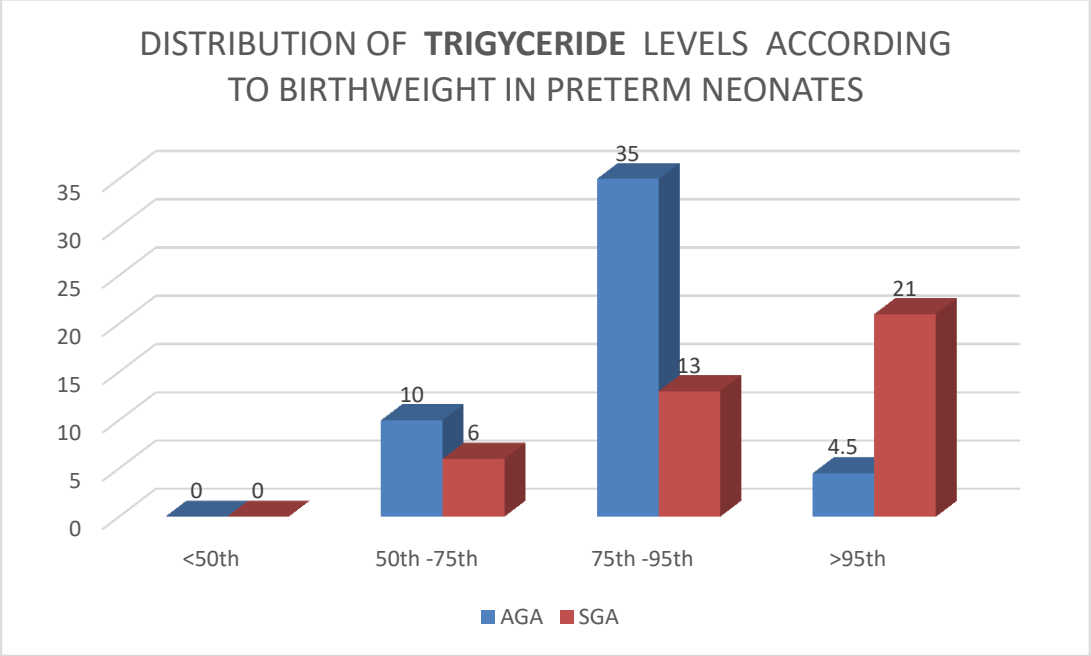
DISTRIBUTION OF TRIGYCERIDE LEVELS ACCORDING TO BIRTHWEIGHT IN TERM NEONATES						
	VALUES (mg/dl)		**GROUP**		**TOTAL**	**P-VALUE**
			AGA	**SGA**		
TRIGYCERIDE LEVELS	<50th (<25)	n	0	0	0	0.0352
		%	0	0	0	
	50^{th} -75^{th} (25 – 92)	n	12	5	17	
		%	12.9%	9.4%	11.6%	
	75^{th} -95^{th} (93-160)	n	41	29	70	
		%	44.0%	54%	47.0%	
	>95^{th} (>160)	n	40	19	59	
		%	43.0%	35.8%	40%	
Total		n	93	53	146	
		%	100%	100%	100%	



Higher percentages of AGA (43%) and SGA (35.8%) term neonates were in the >95th percentile for triglycerides. The difference was significant (p=0.0352).

Table 9 (b) PERCENTILE DISTRIBUTION OF TRIGYCERIDES VALUES AND THEIR COMAPRISON IN AGA AND SGA IN PRETERM NEONATES

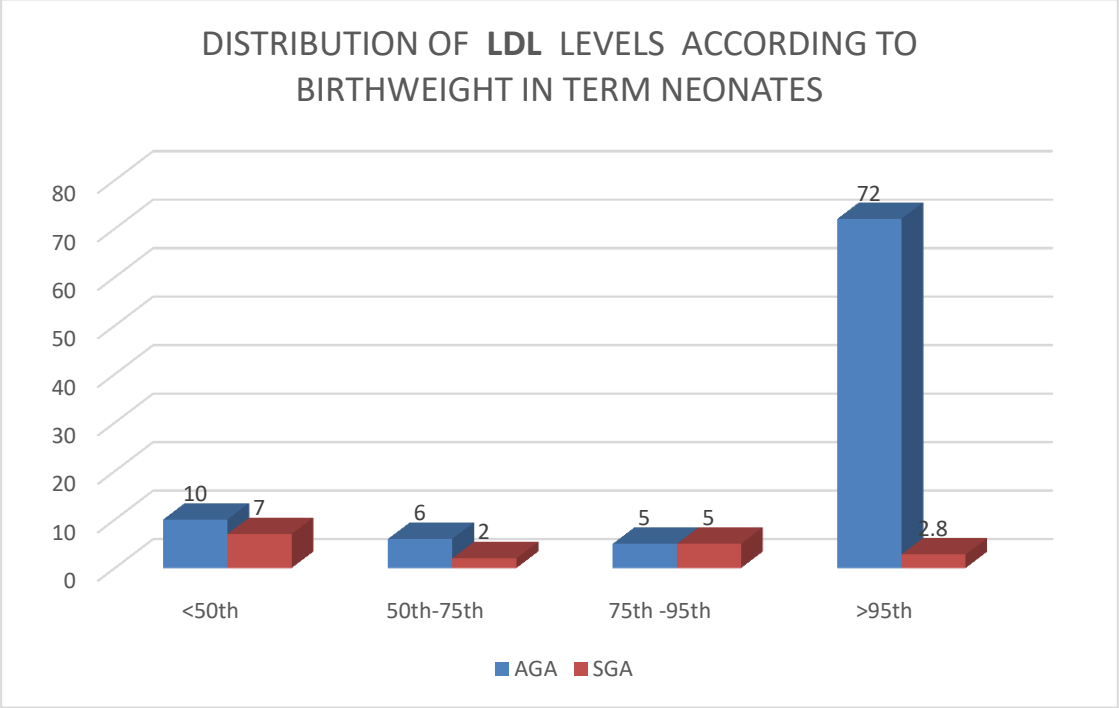
DISTRIBUTION OF TRIGYCERIDE LEVELS ACCORDING TO BIRTHWEIGHT IN PRETERM NEONATES						
	VALUES (mg/dl)		GROUP		TOTAL	P-VALUE
			AGA	SGA		
TRIGYCERIDE LEVELS	<50th (<25)	n	0	0	0	0.102
		%	0%	0%	0%	
	50^{th} -75^{th} (25 – 92)	n	10	6	16	
		%	9.4%	15%	10.9%	
	75^{th} -95^{th} (93-160)	n	35	13	48	
		%	33.0%	32.5%	30.8%	
	>95^{th} (>160)	n	61	21	82	
		%	57.5%	52.5%	56.1%	
Total		n	106	40	146	
		%	100%	100%	100%	



In preterm neonates, more than half of both AGA (57.5%) and SGA (52.5%) had triglyceride levels >160 mg/dL. The difference was not statistically significant (p=0.102).

Table 10 (a) PERCENTILE DISTRIBUTION OF LDLVALUES AND THEIR COMAPRISON IN TERM AGA AND SGA NEONATES

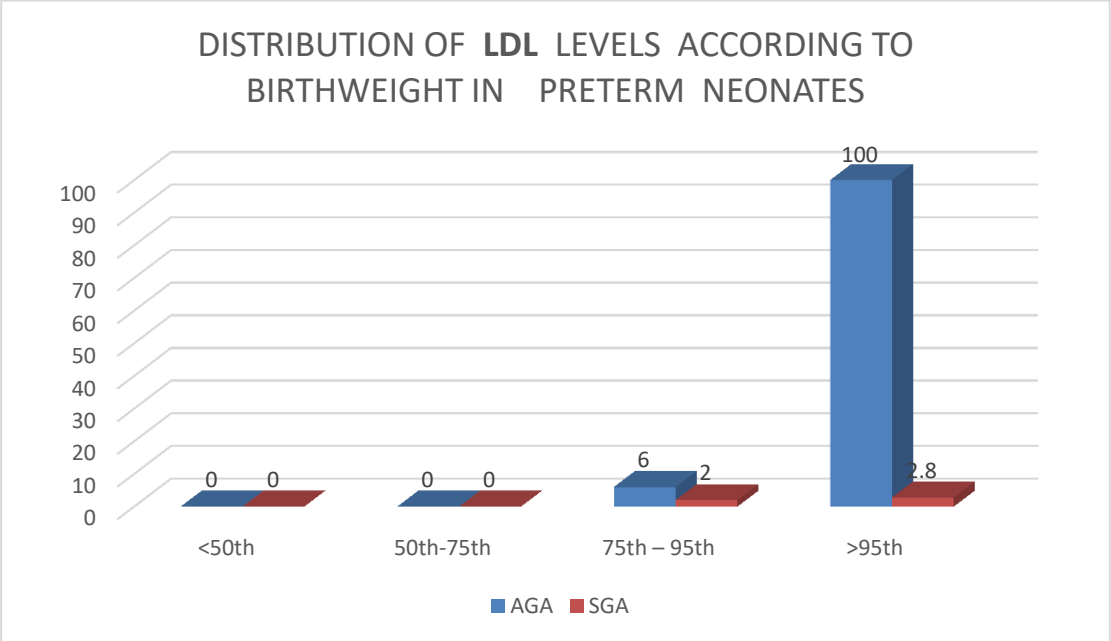
DISTRIBUTION OF LDL LEVELS ACCORDING TO BIRTHWEIGHT IN TERM NEONATES						
	VALUES (mg/dl)		GROUP		TOTAL	P-VALUE
			AGA	SGA		
LDL	<50th (<94)	n	10	7	17	0.0014
		%	10.0%	13.2%	11.6%	
	50th-75th (94-112)	n	6	2	8	
		%	6.4%	3.7%	5.4%	
	75th – 95th (113-130)	n	5	5	10	
		%	5.3%	9.4%	6.8%	
	>95th (>130)	n	72	39	111	
		%	77.4%	73.5%	76.0%	
Total		n	93	53	146	
		%	100%	100%	100%	



A high proportion of both AGA (77.4%) and SGA (73.5%) neonates had LDL >130 mg/dL. However, the distribution across percentiles differed significantly (p=0.0014).

Table 10 (b) PERCENTILE DISTRIBUTION OF LDLVALUES AND THEIR COMAPRISON IN PRETERM AGA AND SGA NEONATES

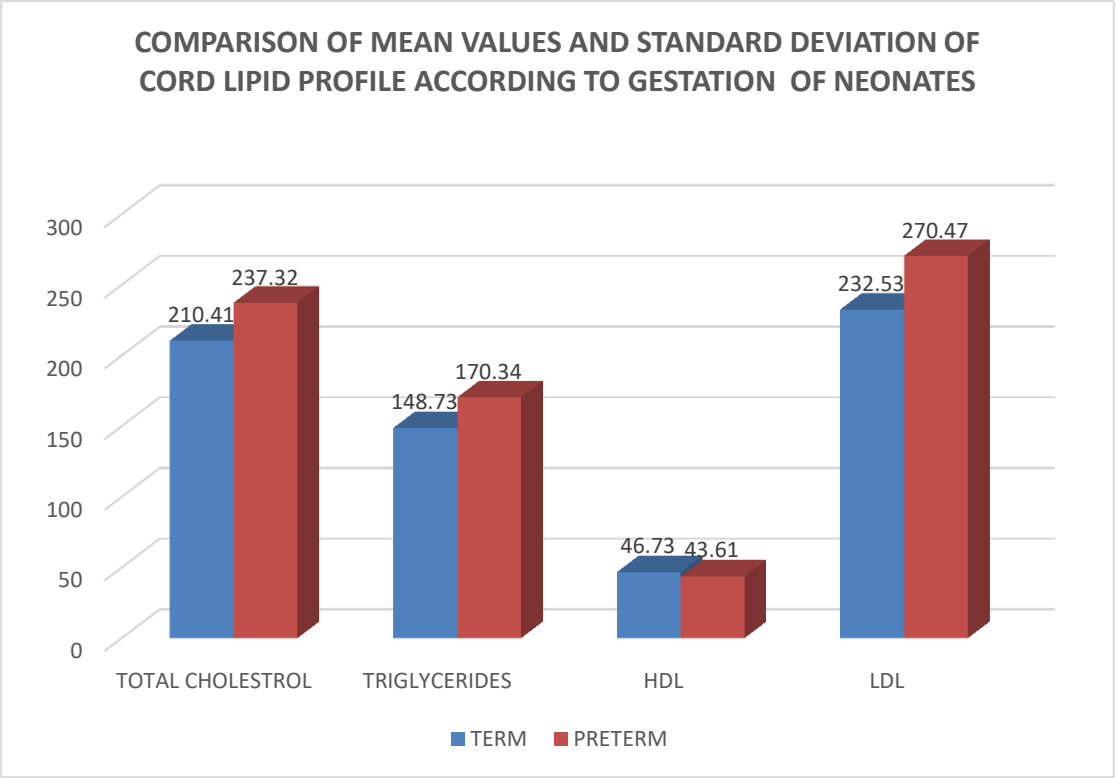
DISTRIBUTION OF LDL LEVELS ACCORDING TO BIRTHWEIGHT IN PRETERM NEONATES						
	VALUES (mg/dl)		GROUP		TOTAL	P- VALUE
			AGA	SGA		
LDL	<50th (<94)	n	0	0	0	0.132
		%	0%	0%	0%	
	50th-75th (94-112)	n	0	0	0	
		%	0%	0%	0%	
	75th – 95th (113-130)	n	6	2	8	
		%	5.6%	5.0%	5.4%	
	>95th (>130)	n	100	38	138	
		%	94.3%	95.0%	94.5%	
Total		n	106	40	146	
		%	100%	100%	100%	



The distribution comparison in preterm AGA (94.3%) and SGA (95%) preterm neonates had LDL >130 mg/dL. No significant difference was noted (p=0.132).

Table 11 COMPARISON OF MEAN VALUES AND STANDARD DEVIATION OF CORD LIPID PROFILE ACCORDING TO GESTATION OF NEONATES *(mg/dl)

LIPID PROFILE *	TERM	PRETERM	P VALUE
TOTAL CHOLESTROL	210.41 ± 77.67	237.32 ± 59.68	0.043
TRIGLYCERIDES	148.73 ± 53.53	170.34 ± 55.03	0.00058
HDL	46.56 ± 12.32	43.61 ± 8.48	0.882
LDL	232.53 ± 111.96	270.47 ± 93.59	0.0053



Preterm neonates had significantly higher total cholesterol (237.32 ± 59.68 vs. 210.41 ± 77.67, p=0.043), triglycerides (170.34 ± 55.03 vs. 148.73 ± 53.53, p=0.00058), and LDL (270.47 ± 93.59 vs. 232.53 ± 111.96, p=0.0053) than term neonates. HDL values were slightly lower in preterm neonates but the difference was not significant (p=0.882).

Table 12 COMPARISON OF MEAN VALUE AND SD OF CORD LIPID PROFILE IN ALL NEONATES ACCORDING TO TERM AGA AND SGA VS PRETERM AGA AND SGA

LIPID PROFILE *	TERM		PRETERM		P VALUE
	AGA	SGA	AGA	SGA	
TOTAL CHOLESTROL	214.32 ± 84.08	203.56 ± 65.13	241.80 ± 56.14	222.45 ± 67.50	0.001
TRIGLYCERIDES	151.90 ± 57.68	143.18 ± 45.34	172.00 ± 52.7	165.95 ± 61.21	0.001
HDL	45.93 ± 12.65	47.67 ± 11.75	43.97 ± 8.37	42.67 ± 8.80	0.321
LDL	241.49 ± 114.34	216.79 ± 106.89	272.09 ± 91.56	266.17 ± 99.84	0.001

*** (mg/dl)**

Preterm AGA and SGA neonates had higher total cholesterol, triglycerides, and LDL levels than their term counterparts. Differences were statistically significant for total cholesterol, triglycerides, and LDL (p=0.001), but not for HDL (p=0.321).

DISCUSSION

DISCUSSION

The study a comparative study of cord blood lipid profile in small for gestational age vs appropriate for gestational age neonates was conducted in the Department of Paediatrics of Santosh medical college and Hospital. The present study examined 292 neonates, with a relatively balanced distribution between term (146 neonates) and preterm (146 neonates). Among term neonates, 63.6% were appropriate for gestational age (AGA) and 36.3% were small for gestational age (SGA), while in preterm neonates, 72.6% were AGA and 27.3% were SGA. This distribution is essential for making meaningful comparisons between these groups and aligns with expected demographic patterns in neonatal populations.

The predominance of AGA neonates in both term and preterm categories is consistent with normal population distributions, while the proportion of SGA neonates in the study cohort allows for robust statistical analysis. Late preterm neonates (32.5%) constituted the largest subgroup, followed by full-term neonates (26.7%), highlighting the importance of understanding the metabolic characteristics of near-term infants who may present with unique physiological challenges.

This distribution pattern differs somewhat from that reported by Ramanathan et al. (2024), whose study included 222 neonates with a different proportion of term (155) to preterm (67) neonates. Their study also included six post-term neonates, a category not represented in the present research. The varying distributions between studies may reflect differences in the populations served by different medical institutions or differences in inclusion criteria.

Machakanur et al. (2022) [85]reported a smaller proportion of SGA neonates (34 out of 133, approximately 25.6%) compared to our study's 31.8% overall SGA rate. This variation could be attributed to different maternal risk factors, socioeconomic conditions, or nutritional status of the populations studied, which are known to influence intrauterine growth.

These differences in distribution patterns across studies emphasize the importance of considering the demographic characteristics of study populations when interpreting

and comparing lipid profile results, as variations in these patterns may influence the overall findings and their clinical implications.

Anthropometric Parameters in Term and Preterm Neonates

The present study demonstrated significant anthropometric differences between SGA and AGA neonates within both term and preterm categories. Term SGA neonates had significantly lower mean weight (2.22 kg vs. 3.08 kg, p=0.0001), head circumference (32.9 cm vs. 34.8 cm, p=0.034), and ponderal index (2.03 vs. 2.85, p=0.0123) compared to Term AGA neonates. Similarly, preterm SGA neonates showed significantly lower weight (1.81 kg vs. 2.29 kg, p=0.0001), head circumference (28.2 cm vs. 30.3 cm, p=0.0342), and ponderal index (2.05 vs. 2.53, p=0.012) compared to preterm AGA neonates. Interestingly, body length did not differ significantly between SGA and AGA neonates in either term (p=0.569) or preterm (p=0.582) groups.

These anthropometric findings align with those reported by Ramanathan et al. (2024), who found significantly lower birth weight in SGA neonates (1.81±0.34 kg) compared to AGA neonates (2.82±0.25 kg) ($p<0.05$). However, unlike the present study, they did not report significant differences in head circumference between SGA and AGA groups. This disparity might be due to differences in the severity of growth restriction in the respective study populations or variations in measurement techniques.

The preservation of body length with reductions in weight and head circumference observed in the present study suggests asymmetrical growth restriction, which typically occurs when intrauterine insults affect later stages of pregnancy. This pattern is often associated with placental insufficiency, resulting in brain-sparing effects where linear growth is relatively preserved while weight gain is compromised. This observation is particularly important as asymmetrical growth restriction has been associated with different metabolic adaptations compared to symmetrical growth restriction, potentially influencing lipid metabolism differently.[84]

The significantly lower ponderal index in SGA neonates in the present study provides further evidence of asymmetrical growth restriction. Among term SGA neonates,

52.8% had a ponderal index <2, and none had an index >2.5, whereas 88.1% of term AGA neonates had a ponderal index >2.5. Similarly, in preterm neonates, 55% of SGA neonates had a ponderal index <2, and none had >2.5, while 52.8% of AGA neonates had a ponderal index >2.5.

Ramanathan et al. (2024) [83] also found a significant correlation between ponderal index and cord blood lipid profiles ($p<0.05$), reinforcing the importance of this parameter in understanding the metabolic consequences of intrauterine growth restriction. Their observation that asymmetrical IUGR neonates had elevated triglycerides and VLDL levels complements our findings and suggests specific metabolic adaptations in response to different patterns of growth restriction.

These anthropometric differences between SGA and AGA neonates provide the foundation for understanding potential variations in lipid metabolism and highlight the importance of considering both birth weight categories and the pattern of growth restriction when evaluating neonatal lipid profiles.

Ponderal Index as an Indicator of Nutritional Status

The ponderal index, calculated as [weight (g) × 100 / length $(cm)^3$], serves as an important indicator of nutritional status and the pattern of intrauterine growth restriction. The present study revealed differences in the distribution of ponderal index between SGA and AGA neonates, emphasizing the nutritional disparities between these groups.

Among term neonates, 52.8% of SGA neonates had a ponderal index <2, indicative of significant wasting and asymmetrical growth restriction, while 47.1% had values between 2-2.5. In stark contrast, 88.1% of term AGA neonates had a ponderal index >2.5, reflecting appropriate nutritional status. Similarly, among preterm neonates, 55% of SGA neonates had a ponderal index <2, and none had values >2.5, whereas 52.8% of AGA neonates had a ponderal index >2.5.

These findings align with the observations of Ramanathan et al. (2024), [83] who reported a significant correlation between ponderal index and cord blood lipid profiles ($p<0.05$). They further distinguished between asymmetrical and symmetrical IUGR

based on ponderal index, noting that asymmetrical IUGR neonates had elevated triglyceride levels (86.70±17.72) and VLDL levels (17.34±3.54) compared to symmetrical IUGR neonates.

The predominance of low ponderal indices in SGA neonates in the present study suggests that most growth-restricted neonates experienced asymmetrical growth restriction, likely resulting from placental insufficiency during the third trimester. This timing allows for relatively normal organ development but compromised adipose tissue deposition, leading to the wasted appearance characteristic of asymmetrical IUGR.

The clinical significance of ponderal index extends beyond its role as a nutritional indicator. Various studies have demonstrated associations between low ponderal index at birth and increased risk of metabolic syndrome, insulin resistance, and cardiovascular disease in later life. [44,48]The "thrifty phenotype" hypothesis proposed by Hales and Barker suggests that intrauterine malnutrition leads to permanent metabolic adaptations that, while beneficial for survival in a nutrient-poor environment, predispose to metabolic disorders when exposed to nutrient abundance later in life.[1]

HDL Cholesterol Distributions

High-density lipoprotein (HDL) cholesterol, often referred to as "good cholesterol," plays a crucial role in reverse cholesterol transport and has protective effects against atherosclerosis. The present study revealed different patterns in HDL levels among different neonatal groups.

In term neonates, a higher proportion of SGA neonates fell into the 75th-95th percentile for HDL (18.8% vs. 9.6% in AGA), with very few below the 50th percentile. This difference was statistically significant (p=0.0001). Conversely, among preterm neonates, 15% of SGA neonates were below the 50th percentile for HDL compared to only 5.6% of AGA neonates, with no neonates above the 95th percentile. This distribution was also statistically significant (p=0.0001).

These findings present a complex picture that differs from some previous reports.

Ramanathan et al. (2024) [83] found that SGA neonates had lower HDL levels (32.77±09.57) compared to AGA neonates, though the specific percentile distributions were not reported. Similarly, Arora et al. (2017)[87] observed slightly lower HDL in SGA neonates (27.70±7.79 mg/dL) compared to AGA neonates (28.51±5.68 mg/dL), although this difference was not statistically significant.

The higher HDL levels in term SGA neonates in the present study might represent a compensatory mechanism to counterbalance the generally adverse lipid profile associated with intrauterine growth restriction. This could be viewed as a protective adaptation, potentially mitigating the atherogenic risk conferred by elevated levels of other lipid fractions.

However, the lower HDL levels in preterm SGA neonates compared to their AGA counterparts align with findings from Amandeep et al. (2019), [86]who reported that HDL levels were significantly lower in preterm neonates compared to term neonates. This suggests that prematurity may compromise the compensatory increase in HDL observed in term SGA neonates, potentially exacerbating their cardiovascular risk profile.

Total Cholesterol

Total cholesterol levels provide a comprehensive measure of cholesterol status and are an important component of the lipid profile assessment. The present study revealed significant differences in total cholesterol distributions between SGA and AGA neonates, with distinct patterns in term and preterm groups.

In term neonates, AGA neonates showed a higher proportion in the >95th percentile for total cholesterol (36.3%) compared to SGA neonates (26.4%), with significant differences in overall distribution (p=0.0021). Similarly, in preterm neonates, a greater proportion of AGA neonates had total cholesterol >250 mg/dL (49%) compared to SGA neonates (35%), a difference that was also statistically significant (p=0.0001).

These findings contrast with several previous studies. Ramanathan et al. (2024) [83] reported higher total cholesterol in preterm neonates compared to term neonates, and

in SGA neonates compared to AGA neonates. Mantry et al. (2024) found significantly higher levels of serum total cholesterol in SGA neonates compared to AGA neonates. Similarly, Machakanur et al. (2022) [85] reported higher mean total cholesterol in SGA neonates (63.62±40.48 mg/dL) compared to AGA neonates (48.69±2.29 mg/dL, p=0.007), and Arora et al. (2017) [87] observed higher total cholesterol in SGA neonates (100.85±26.82 mg/dL) compared to AGA neonates (72.29±21.13 mg/dL).

The divergent findings in the present study, with AGA neonates showing higher proportions in the upper percentiles of total cholesterol, are intriguing and warrant further investigation. These differences might be attributed to variations in maternal factors such as diet, lipid metabolism, or placental function, which can significantly influence fetal lipid levels. Additionally, differences in the criteria for defining SGA or in the severity of growth restriction between studies may contribute to these discrepancies.

Despite these differences, the overall finding that preterm neonates had significantly higher mean total cholesterol (237.32 ± 59.68 mg/dL) compared to term neonates (210.41 ± 77.67 mg/dL, p=0.043) aligns with observations from Ramanathan et al. (2024) [83] and Amandeep et al. (2019), [86] who also reported higher cholesterol levels in preterm neonates. This consistency suggests that gestational age is an important determinant of neonatal cholesterol levels, potentially due to the developmental trajectory of hepatic function and cholesterol metabolism.

Triglyceride Distributions

Triglycerides represent an important energy storage form and are a key component of the lipid profile. The present study revealed interesting patterns in triglyceride distributions across different neonatal groups.

In term neonates, higher percentages of both AGA (43%) and SGA (35.8%) neonates were in the >95th percentile for triglycerides, with significant differences in the overall distribution (p=0.0352). In preterm neonates, more than half of both AGA (57.5%) and SGA (52.5%) had triglyceride levels >160 mg/dL, although the difference in distribution was not statistically significant (p=0.102).

These findings partially align with previous studies. Ramanathan et al. (2024) [83] reported higher triglyceride levels in preterm neonates (80.75±12.84 mg/dL) compared to term neonates (71.21±22.35 mg/dL), similar to the observation in the present study that preterm neonates had significantly higher mean triglycerides (170.34 ± 55.03 mg/dL) compared to term neonates (148.73 ± 53.53 mg/dL, p=0.00058).

However, Ramanathan et al. (2024) [83] also found higher triglyceride levels in asymmetrical IUGR neonates (86.70±17.72 mg/dL) compared to symmetrical IUGR neonates (71.38±21.86 mg/dL). Mantry et al. (2024), [84] Arora et al. (2017), [87] and Lobo et al. (2016) [88] all reported significantly higher triglyceride levels in SGA neonates compared to AGA neonates. These findings contrast with the present study observation that AGA term neonates had a slightly higher proportion in the >95th percentile for triglycerides compared to SGA term neonates.

The discrepancy might be explained by various factors. First, the present study reports distributions across percentiles rather than mean values, potentially highlighting patterns at the extremes of the distribution that might not be apparent in comparisons of means. Second, maternal factors such as diet, metabolic status, and pregnancy complications can significantly influence fetal triglyceride levels and may vary between study populations. Third, differences in the definition and severity of growth restriction between studies may contribute to the observed variations.

The notably high proportion of preterm neonates with elevated triglycerides in the present study (over 50% in both AGA and SGA groups having levels >160 mg/dL) aligns with the general observation across multiple studies that prematurity is associated with higher triglyceride levels. This may reflect the incomplete development of hepatic enzymes involved in triglyceride metabolism in preterm infants, as well as potential adaptations to the metabolic challenges of premature birth.

LDL Cholesterol

Low-density lipoprotein (LDL) cholesterol, often termed "bad cholesterol," is a critical component of the lipid profile with significant implications for future

cardiovascular risk. In the present study revealed remarkably high LDL levels across all neonatal groups, with some notable patterns of distribution.

In term neonates, a high proportion of both AGA (77.4%) and SGA (73.5%) neonates had LDL >130 mg/dL, though the distribution across percentiles differed significantly (p=0.0014). Even more striking was the observation that nearly all preterm neonates, both AGA (94.3%) and SGA (95%), had LDL >130 mg/dL, with no significant difference in distribution between these groups (p=0.132).

These findings reveal a prevalence of elevated LDL levels in neonates, particularly in the preterm population. The threshold of 130 mg/dL is considered the upper limit of normal for adults according to many guidelines, and the fact that the vast majority of neonates in the present study exceeded this level warrants attention.

Comparison with previous studies reveals both consistencies and variations. Ramanathan et al. (2024) [83] reported higher LDL in preterm neonates (53.77±25.25 mg/dL) compared to term neonates (39.85±14.10 mg/dL), and in SGA neonates (48.57±22.88 mg/dL) compared to AGA neonates (40.75±15.66 mg/dL). Mantry et al. (2024) [84] similarly found significantly higher LDL in SGA neonates compared to AGA neonates. Arora et al. (2017) [87] reported higher LDL in SGA neonates (44.93±7.66 mg/dL) compared to AGA neonates (34.94±16.45 mg/dL), and Lobo et al. (2016) [88]also observed significantly higher LDL in SGA neonates compared to AGA neonates.

While these studies consistently report higher LDL in SGA compared to AGA neonates, the present study found a slightly higher proportion of AGA term neonates with LDL >130 mg/dL compared to SGA term neonates. However, this pattern was reversed in the preterm group, with a marginally higher proportion of SGA neonates having LDL >130 mg/dL.

The most striking aspect of the present study was the findings in the absolute LDL levels, which appear substantially higher than those reported in previous studies. Our comparison of mean values reveals that preterm neonates had significantly higher mean LDL (270.47 ± 93.59 mg/dL) compared to term neonates (232.53 ± 111.96

mg/dL, p=0.0053). These values are multiple times higher than those reported by Ramanathan et al. (2024), [83] Arora et al. (2017), and other studies.[87,88]

Comparison of Mean Lipid Values Between Groups

The present study comprehensive analysis of mean lipid values revealed significant differences between term and preterm neonates, as well as between AGA and SGA neonates within these categories.

Preterm AGA and SGA neonates had higher total cholesterol, triglycerides, and LDL levels than their term counterparts. These differences were statistically significant for total cholesterol, triglycerides, and LDL (p=0.001), but not for HDL (p=0.321). When comparing all term versus all preterm neonates, preterm neonates had significantly higher total cholesterol (237.32 ± 59.68 vs. 210.41 ± 77.67 mg/dL, p=0.043), triglycerides (170.34 ± 55.03 vs. 148.73 ± 53.53 mg/dL, p=0.00058), and LDL (270.47 ± 93.59 vs. 232.53 ± 111.96 mg/dL, p=0.0053). HDL values were slightly lower in preterm neonates, but this difference was not statistically significant (p=0.882).

These findings align with the observations of Ramanathan et al. (2024),[83] who reported higher cord lipid profile values in preterm neonates compared to term neonates for total cholesterol (98.91±26.60 vs. 82.63±27.16 mg/dL), triglycerides (80.75±12.84 vs. 71.21±22.35 mg/dL), LDL (53.77±25.25 vs. 39.85±14.10 mg/dL), and VLDL (16.15±04.30 vs. 14.43±04.47 mg/dL). Amandeep et al. (2019) [86] similarly found that preterm neonates had higher levels of total cholesterol, LDL, VLDL, and triglycerides compared to term neonates.

The higher lipid levels in preterm neonates might be attributed to several factors. The liver, which plays a central role in lipid metabolism, continues to develop throughout gestation, with many metabolic pathways not fully functional until late gestation or even after birth. Preterm neonates may therefore have reduced capacity for lipid clearance and metabolism. Additionally, the stress of premature birth and associated complications may trigger hormonal responses that affect lipid metabolism. The higher metabolic demands and nutritional challenges faced by preterm infants might

also necessitate adaptations in lipid metabolism to ensure adequate energy supply.

Regarding the comparison between SGA and AGA neonates, the present study findings revealed a complex picture. While most previous studies have consistently reported higher lipid levels in SGA neonates, the results suggest a more nuanced relationship that varies depending on gestational age and specific lipid fractions. This complexity underscores the multifactorial nature of neonatal lipid metabolism and the importance of considering both gestational age and growth status when evaluating neonatal lipid profiles.

CONCLUSION AND RECOMMENDATIONS

CONCLUSION & RECOMMENDATIONS

Conclusion:

This study demonstrates that gestational age and intrauterine growth status substantially influence cord blood lipid profiles in neonates. Preterm birth is associated with a more atherogenic lipid profile, characterized by significantly elevated total cholesterol, triglycerides, and LDL levels compared to term birth. The relationship between growth status and lipid profiles is more complex and varies by gestational age and specific lipid fractions.

The remarkably high prevalence of elevated LDL levels, particularly in preterm neonates, raises concerns about future cardiovascular risk. Nearly all preterm neonates (94-95%) had LDL levels exceeding 130 mg/dL, a threshold considered the upper limit of normal for adults by many guidelines. The anthropometric findings, especially the high prevalence of low ponderal indices in SGA neonates, indicate asymmetrical growth restriction predominated in our cohort. This pattern, typically resulting from third-trimester placental insufficiency, may represent a distinct metabolic phenotype with specific implications for lipid metabolism. The complex interplay between gestational age, growth status, and lipid metabolism underscores the importance of individualized risk assessment and follow-up for preterm and SGA neonates, with potential implications for lifelong cardiovascular health management.

Strengths

1. Large, balanced sample size: The study included 292 neonates with equal distribution between term and preterm groups (146 each), providing robust statistical power for comparisons.
2. Comprehensive lipid profile assessment: Analysis of multiple lipid fractions (total cholesterol, HDL, LDL, and triglycerides) offered a complete picture of neonatal lipid metabolism.

3. Detailed anthropometric measurements: Collection of weight, length, head circumference, and calculation of ponderal index allowed thorough characterization of growth patterns and nutritional status.
4. Percentile-based analysis: Reporting distributions across percentiles rather than just mean values revealed patterns at the extremes of distributions that might otherwise be obscured.
5. Ponderal index assessment: Inclusion of this parameter provided insights into the pattern of growth restriction (symmetrical vs. asymmetrical) and its potential metabolic implications.

Limitations

1. Limited maternal data: The study collected minimal information on maternal factors beyond gravidity and delivery mode, restricting analysis of maternal influences on fetal lipid metabolism.
2. Cross-sectional design: The absence of longitudinal follow-up prevented assessment of the persistence of lipid abnormalities and their clinical outcomes.

Recommendations

1. Implement routine cord blood lipid screening for preterm and SGA neonates to identify those with particularly concerning profiles for targeted follow-up.
2. Develop longitudinal monitoring protocols for preterm and SGA neonates, with regular assessment of lipid profiles and other cardiovascular risk factors throughout childhood and adolescence.
3. Prioritize breastfeeding support for preterm and SGA infants, given its potential protective effects against adverse metabolic programming.
4. Establish nutritional guidelines specific to preterm and SGA infants that consider their unique metabolic profiles and long-term cardiovascular health.

5. Create integrated care pathways involving neonatologists, pediatricians, and pediatric cardiologists for high-risk neonates identified through cord blood lipid screening.

6. Conduct longitudinal studies tracking lipid profiles from birth through childhood and into adulthood to determine the persistence of abnormalities and their clinical significance.

SUMMARY

SUMMARY

Summary;

This comparative study analysed cord blood lipid profiles in 292 neonates (146 term and 146 preterm), examining differences between small for gestational age (SGA) and appropriate for gestational age (AGA) infants.

- Among term neonates, 63.6% were AGA and 36.3% were SGA, while preterm neonates included 72.6% AGA and 27.3% SGA.

- Significant anthropometric differences were observed between SGA and AGA neonates in both gestational age groups. Term SGA neonates had lower mean weight (2.22 kg vs. 3.08 kg), head circumference (32.9 cm vs. 34.8 cm), and ponderal index (2.03 vs. 2.85) compared to term AGA neonates. Similar differences were found in preterm neonates. The ponderal index distribution revealed that 52.8% of term SGA neonates and 55% of preterm SGA neonates had values <2, indicating asymmetrical growth restriction.
- In term neonates, SGA infants had higher proportions in the 75th-95th percentile for HDL (18.8% vs. 9.6% in AGA). Conversely, AGA term neonates showed higher proportions in the >95th percentile for total cholesterol (36.3% vs. 26.4% in SGA). For LDL, high levels (>130 mg/dL) were prevalent in all groups but particularly in preterm neonates (94.3% of AGA and 95% of SGA).
- When comparing all term versus preterm neonates, preterm infants had significantly higher mean values for total cholesterol (237.32 vs. 210.41 mg/dL), triglycerides (170.34 vs. 148.73 mg/dL), and LDL (270.47 vs. 232.53 mg/dL). HDL values were slightly lower in preterm neonates but not significantly different.
- These findings suggest that both prematurity and intrauterine growth restriction significantly impact neonatal lipid metabolism, with potential implications for future cardiovascular risk.

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ANNEXURES

PROFORMA (ANNEXURE 1)

CASE RECORD FORM

TITLE: A Comparative study of cord blood lipid profile in small for gestational age vs appropriate for gestational age neonates

.

PATIENT DETAILS

Name: B/O Name of Mother:

Date of Birth and Time of Birth: Place of Birth:

Date and Time of Sample Collection:

Gender: 1. M / 2. F

Address:

MATERNAL HISTORY

Family History of: HTN / DM / CHD / Hypercholesterolemia: 1. Yes, 2. No

Paternal History of:

Parity: 1. Primi, 2. G2, 3. G3, 4. ≥G4

ANC Check-up: 1. Regular, 2. Irregular, 3. Not taken

ANC H/O LMP: EDD:

PROBLEMS DURING PREGNANCY (PDP)

1. No Problems. 2. Anaemia 3. PIH 4. Pre-eclampsia. 5. Eclampsia

6. APH 7. UTI 8. RHD / CHD 9. Fever 10.

Others:

NATAL HISTORY

Mode of Delivery (MOD): 1. Normal vaginal delivery, 2. C-section 3. Others

Birth Asphyxia: 1. Present, 2. Absent

Apgar Score: 1 min: _____, 5 min: _____

Intervention after Delivery (IAD):

1. Not done 2. Stimulation and suctioning 3. Bag & mask ventilation

4. ET tube ventilation

GENERAL PHYSICAL EXAMINATION

1. Normal 2. Abnormal

ANTHROPOMETRY

Weight: _____ Kg (SGA / AGA / LGA)

Head Circumference (HC): _____ cm

Length: _____ cm

Ponderal Index: _____ gm/cm³

VITAL SIGNS

Heart Rate: _____ / min

Respiratory Rate: _____ / min

Temperature: _____

Capillary Refilling Time: <3 sec / >3 sec

HEAD TO TOE EXAMINATION

- Anterior Fontanelle (AF)
- Eyes
- Ear/Nose
- Oral Cavity
- Neck
- Chest
- Abdomen, Umbilical Cord
- Hip
- Extremities
- Skin
- Back, Anus, Rectal Exam
- Genitalia
- NBR (Neonatal Behavioral Reflexes) - Rooting, Suckling, Moro's Reflex

SYSTEMIC EXAMINATION

CVS: 1. Normal, 2. Abnormal

RS: 1. Normal, 2. Abnormal

PA: 1. Normal, 2. Abnormal

CNS: 1. Normal, 2. Abnormal

LIPID PROFILE

Total Cholesterol (TC):

Triglycerides (TGL):

HDL Cholesterol (HDL):

LDL Cholesterol (LDL):

VLDL Cholesterol (VLDL):

Atherogenic Index (AI)

Signature of Investigator: _____
Date: _____

ANNEXURE II

INFORMED CONSENT FORM

Title: of research project - A Comparative Study of Cord Blood Lipid Profile in Small for Gestational Age vs. Appropriate for Gestational Age Neonates

Participant Information

I have been informed that your child named _____with mothers name _____and age (In years)_____ would be participating in the research study titled "A Comparative Study of Cord Blood Lipid Profile in Small for Gestational Age vs. Appropriate for Gestational Age Neonates" conducted at [Santosh hospital]. I have read and understood the details mentioned in the Patient Information Sheet.

Consent Statement

-My participation in this study is voluntary, and I can withdraw at any time without affecting my baby's medical care.

The purpose of the study is to analyze the lipid profile in the umbilical cord blood of my newborn.

In this study only common investigations will be done and not add any financial burden on the family

The sample collection will be performed immediately after birth and does not require any additional invasive procedures on my baby.

There are no anticipated risks to my baby due to participation in this study.

My personal and medical data will be kept confidential and used only for research and academic purposes.

Declaration by the Parent/Guardian

I, [Name], the parent/guardian of B/O [Mother's Name], hereby give my informed consent to participate in this study. I have been given the opportunity to ask questions, and my queries have been satisfactorily answered.

Signature of Parent/Guardian: _____

Date: _____

Declaration by the Investigator

I, [Investigator's Name], have explained the study details to the participant, addressed their questions, and ensured they understand the study's purpose and procedures. I confirm that no coercion or undue influence was used to obtain consent.

Signature of Investigator: _____

Date: _____

Witness (if applicable)

Signature of Witness: _____

Date: _____

अधिसूचित सहमति प्रपत्र

शीर्षक: छोटे गर्भकालीन उम्र (SGA) और उपयुक्त गर्भकालीन उम्र (AGA) नवजात शिशुओं में नाल रक्त लिपिड प्रोफ़ाइल का तुलनात्मक अध्ययन।

सहमति कथन

- इस अध्ययन में मेरी भागीदारी स्वैच्छिक है और मैं किसी भी समय बिना किसी दंड के बाहर निकल सकता/सकती हूँ।
- इस अध्ययन का उद्देश्य मेरे नवजात शिशु के नाल रक्त में लिपिड प्रोफ़ाइल का विश्लेषण करना है।
- यह नमूना जन्म के तुरंत बाद लिया जाएगा और मेरे शिशु पर कोई अतिरिक्त आक्रामक प्रक्रिया की आवश्यकता नहीं होगी।
- इस अध्ययन में भाग लेने से मेरे शिशु को किसी भी प्रकार का कोई संभावित खतरा नहीं होगा।
- मेरी व्यक्तिगत और चिकित्सीय जानकारी को गोपनीय रखा जाएगा और केवल शैक्षणिक और अनुसंधान प्रयोजनों के लिए उपयोग किया जाएगा।

अभिभावक/संरक्षक की घोषणा

मैं, [नाम], बी/ओ [माता का नाम] का अभिभावक/संरक्षक, इस अध्ययन में भाग लेने के लिए अपनी सूचित सहमति देता/देती हूँ। मुझे प्रश्न पूछने का अवसर प्रदान किया गया है, और मेरे सभी प्रश्नों के संतोषजनक उत्तर दिए गए हैं।

अभिभावक/संरक्षक के हस्ताक्षर: _____

तारीख: _____

अन्वेषक की घोषणा

मैं, [अन्वेषक का नाम], ने प्रतिभागी को अध्ययन का विवरण समझाया है, उनकी सभी शंकाओं का समाधान किया है और सुनिश्चित किया है कि वे अध्ययन के उद्देश्य और प्रक्रियाओं को समझते हैं। मैं पुष्टि करता/करती हूँ कि सहमति प्राप्त करने के लिए किसी भी प्रकार का दबाव या प्रभाव नहीं डाला गया है।

अन्वेषक के हस्ताक्षर: _____

तारीख: _____

गवाह (यदि लागू हो)

गवाह के हस्ताक्षर: _____

तारीख: _____

Annexure III

List of Abbreviations

Abbreviation	Full Form
AGA	Appropriate for Gestational Age
SGA	Small for Gestational Age
LBW	Low Birth Weight
VLBW	Very Low Birth Weight
ELBW	Extremely Low Birth Weight
NICU	Neonatal Intensive Care Unit
HDL	High-Density Lipoprotein
LDL	Low-Density Lipoprotein
VLDL	Very Low-Density Lipoprotein
TC	Total Cholesterol
TG	Triglycerides
BMI	Body Mass Index
PI	Ponderal Index
GA	Gestational Age
LGA	Large for Gestational Age
SD	Standard Deviation
CI	Confidence Interval
OR	Odds Ratio
RR	Relative Risk
IUGR	Intrauterine Growth Restriction
NEC	Necrotizing Enterocolitis

ROP	Retinopathy of Prematurity
CPAP	Continuous Positive Airway Pressure
G2	Gravida 2 (Second Pregnancy)
G3	Gravida 3 (Third Pregnancy)
G4	Gravida 4 (Fourth Pregnancy)
ANC Visit	Antenatal Care Visit
HTN	Hypertension
DM	Diabetes Mellitus
CHD	Congenital Heart Disease / Coronary Heart Disease (depending on context)
LMP	Last Menstrual Period
EDD	Estimated Due Date
UTI	Urinary Tract Infection
RHD	Rheumatic Heart Disease
HC	Head Circumference
NBR	Neonatal Birth Record

ANTI PLAGIARISM CERTIFICATE

Arpit Kaur

A COMPARITIVE STUDY OF CORD BLOOD LIPID PROFILE IN SMALL FOR GESTATIONAL AGE VS APPROPRIATE FOR GEST

Santosh University, Ghaziabad

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F. No. SU/R/2023/2489[30] Dated: 26.09.2023

Subject: Grant of Ethical Clearance for **Thesis Protocol** Topic **"A Comparative study of cord blood lipid profile in small for gestational age vs appropriate for gestational age neonates",** submitted by **Dr. ARPIT KAUR BHATIA,** MD/MS student of Batch 2022-2023, Department of **PAEDIATRICS.**

*****

With reference to his/her request for grant of Ethical Clearance for the Thesis Protocol entitled **"A Comparative study of cord blood lipid profile in small for gestational age vs appropriate for gestational age neonates", Dr. ARPIT KAUR BHATIA** is informed that the **Thesis Protocol** submitted by him/her was considered by the **Screening Committee** of the Santosh Medical College & Hospital in its meetings held on 17.07.2023 to 28.07.2023. The recommendations of the **Screening Committee** were considered in detail by the **Institutional Ethics Committee** in its meeting held on **23.09.2023** and the same was **approved** by the **Ethics Committee.**

He /She is informed accordingly for further necessary action.

[Dr. NARESH SHARMA]
REGISTRAR

REGISTRAR

Dr. ARPIT KAUR BHATIA
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16th February 2025

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Vice Chancellor

MASTER CHART

MASTER CHART

Registration ID	GROUP	Age of mother	GRAVIDA	POG	Mode of delivery	Birth weight	Gender	Length	HC	PI	Sga/Aga	S.LDL	S. Total cholosterol	S.HDL levels	S.Triglyceride levels
IP250401010	TERM	25	1	37	Vaginal delivery	2300	Female	49	33	1.95	SGA	81	106	33	174
IP250401011	TERM	22	2	39	Cesarean section	2300	Female	49	33	1.95	SGA	178	241	51	36
IP250401012	TERM	20	1	39	Vaginal delivery	1900	Male	47	32	1.83	SGA	413	224	49	128
IP250401013	TERM	24	1	37	Cesarean section	2200	Female	49	32	1.87	SGA	308	316	42	257
IP250401014	TERM	32	3	37	Vaginal delivery	2850	Male	47	35	2.75	AGA	365	246	43	164
IP250401015	TERM	27	2	38	Cesarean section	2000	Male	48	32.5	1.81	SGA	368	306	42	230
IP250401016	TERM	23	2	40	Vaginal delivery	2080	Female	48	32.5	1.88	SGA	160	83	35	160
IP250401017	TERM	27	2	38	Vaginal delivery	3100	Male	49	35.5	2.63	AGA	178	140	43	56
IP250401018	TERM	24	2	37	Cesarean section	2100	Female	48	31	1.90	SGA	335	230	42	140
IP250401019	TERM	22	2	40	Vaginal delivery	2400	Female	50	35	1.92	SGA	119	122	41	167
IP250401020	TERM	22	3	41	Vaginal delivery	2650	Male	51	35	2.00	SGA	119	122	41	167
IP250401021	TERM	22	1	38	Vaginal delivery	2770	Male	46	35.5	2.85	AGA	89	131	40	165
IP250401022	TERM	21	1	39	Vaginal delivery	3520	Female	48	36	3.18	AGA	88	110	60	120
IP250401023	TERM	40	2	39	Vaginal delivery	3000	Male	47	36	2.89	AGA	251	250	42	130
IP250401024	TERM	21	2	39	Cesarean section	2710	Female	45	36	2.97	AGA	191	254	42	167
IP250401025	TERM	24	2	39	Cesarean section	2750	Female	46.5	36	2.74	AGA	363	260	40	200
IP250401026	TERM	25	1	40	Vaginal delivery	3820	Female	48	36.5	3.45	AGA	487	291	43	166
IP250401027	TERM	25	1	40	Vaginal delivery	2000	Female	49	34.5	1.70	SGA	172	191	42	118
IP250401028	TERM	20	1	41	Vaginal delivery	2520	Female	47	34	2.43	SGA	195	214	36	128

IP250401029	TERM	23	2	38	Cesarean section	3000	Male	47	35.5	2.89	AGA	318	209	34	150
IP250401030	TERM	25	1	40	Vaginal delivery	2830	Female	47.5	36.5	2.64	AGA	299	231	65	177
IP250401031	TERM	19	1	39	Vaginal delivery	3270	Male	48.5	36	2.87	AGA	258	221	42	129
IP250401032	TERM	25	1	40	Vaginal delivery	2620	Female	51	34.5	1.98	SGA	182	262	42	154
IP250401033	TERM	25	2	37	Cesarean section	2820	Male	45	35	3.09	AGA	105	106	41	71
IP250401034	TERM	20	1	38	Cesarean section	2900	Female	48	34.5	2.62	AGA	195	285	44	216
IP250401035	TERM	28	2	39	Vaginal delivery	3920	Female	50	36	3.14	AGA	409	374	58	228
IP250401036	TERM	29	3	41	Vaginal delivery	2000	Male	46	33	2.05	SGA	487	291	43	166
IP250401037	TERM	20	1	38	Cesarean section	2930	Female	47.5	34.5	2.73	AGA	266	146	40	100
IP250401038	TERM	20	1	38	Cesarean section	2930	Female	45.5	35.5	3.11	AGA	322	302	40	207
IP250401039	TERM	22	2	37	Vaginal delivery	2620	Female	46	34	2.69	AGA	308	247	40	154
IP250401040	TERM	25	2	38	Vaginal delivery	2440	Male	47	31.5	2.35	SGA	299	231	65	177
IP250401041	TERM	30	3	39	Vaginal delivery	2800	Male	46.5	34	2.78	AGA	103	128	38	90
IP250401042	TERM	22	2	37	Vaginal delivery	2800	Male	46.5	33	2.78	AGA	102	120	65	120
IP250401043	TERM	24	2	38	Cesarean section	2850	Male	50	33.5	2.28	AGA	203	258	43	179
IP250401044	TERM	23	2	40	Vaginal delivery	2750	Female	45	33.5	3.02	AGA	477	270	43	185
IP250401045	TERM	22	1	41	Vaginal delivery	3440	Male	49.5	37	2.84	AGA	372	240	41	176
IP250401046	TERM	26	3	39	Cesarean section	3460	Male	50	36	2.77	AGA	142	189	41	126
IP250401047	TERM	21	1	40	Vaginal delivery	2600	Female	48	33.5	2.35	SGA	117	163	43	116
IP250401048	TERM	22	1	38	Vaginal delivery	2350	Female	47	31.5	2.26	SGA	193	177	43	124
IP250401049	TERM	28	3	38	Vaginal delivery	3370	Female	50	35.5	2.70	AGA	283	244	45	142
IP250401050	TERM	24	1	38	Cesarean section	3200	Female	51	33.5	2.41	AGA	122	130	80	120
IP250401051	TERM	28	1	37	Vaginal delivery	2900	Male	48	32	2.62	AGA	200	201	44	115
IP250401052	TERM	25	2	38	Cesarean section	1900	Female	43	31.5	2.39	SGA	228	248	40	181
IP250401053	TERM	24	2	39	Vaginal delivery	2340	Female	46	32	2.40	SGA	123	130	60	110
IP250401054	TERM	19	1	37	Vaginal delivery	2300	Male	49	32	1.95	SGA	145	189	52	102

IP250401055	TERM	26	2	39	Cesarean section	2100	Female	48	32	1.90	SGA	278	293	36	99
IP250401056	TERM	24	2	37	Cesarean section	1770	Female	42	31	2.39	SGA	174	275	43	139
IP250401057	TERM	21	1	41	Cesarean section	2420	Female	50	33	1.94	SGA	140	195	44	133
IP250401058	TERM	30	4	38	Cesarean section	3090	Male	47.5	33.5	2.88	AGA	82	106	36	69
IP250401059	TERM	24	1	39	Vaginal delivery	2995	Female	52	35	2.13	AGA	262	307	45	99
IP250401060	TERM	21	2	39	Vaginal delivery	3220	Female	47	36	3.10	AGA	264	253	41	212
IP250401061	TERM	28	2	40	Vaginal delivery	2850	Female	46	34.5	2.93	AGA	140	120	64	120
IP250401062	TERM	27	2	39	Vaginal delivery	2980	Male	45.5	36	3.16	AGA	131	118	39	78
IP250401063	TERM	21	2	38	Vaginal delivery	3200	Female	48	35.5	2.89	AGA	432	279	55	194
IP250401064	TERM	30	2	38	Vaginal delivery	3560	Male	50	35.5	2.85	AGA	168	256	42	227
IP250401065	TERM	19	1	40	Vaginal delivery	3180	Female	54	34.5	2.02	AGA	228	175	39	126
IP250401066	TERM	22	1	40	Vaginal delivery	2710	Male	47	35.5	2.61	AGA	412	232	35	179
IP250401067	TERM	20	1	40	Vaginal delivery	1800	Female	45	32.5	1.98	SGA	110	140	70	110
IP250401068	TERM	22	2	39	Vaginal delivery	3110	Male	49	34	2.64	AGA	200	213	25	157
IP250401069	TERM	24	1	39	Vaginal delivery	3500	Male	48	33	3.16	AGA	205	200	41	131
IP250401070	TERM	28	2	39	Cesarean section	3380	Male	48	36	3.06	AGA	146	228	55	166
IP250401071	TERM	28	2	40	Vaginal delivery	1900	Female	44	32.5	2.23	SGA	341	277	64	208
IP250401072	TERM	27	1	38	Vaginal delivery	2400	Male	49.5	32.5	1.98	SGA	81	116	43	65
IP250401073	TERM	23	2	39	Vaginal delivery	2890	Female	47	35	2.78	AGA	139	165	46	76
IP250401074	TERM	35	2	37	Vaginal delivery	2900	Female	46	34	2.98	AGA	435	329	55	254
IP250401075	TERM	27	1	40	Vaginal delivery	3900	Male	50	36.5	3.12	AGA	287	446	69	297
IP250401076	TERM	31	1	38	Vaginal delivery	1500	Male	44	31.5	1.76	SGA	151	211	56	146
IP250401077	TERM	28	2	37	Vaginal delivery	2500	Male	45	31	2.74	AGA	316	243	67	178
IP250401078	TERM	20	1	37	Vaginal delivery	2600	Male	47	33	2.50	AGA	234	268	46	191
IP250401079	TERM	27	2	38	Vaginal delivery	2850	Male	46	34.5	2.93	AGA	322	351	56	259
IP250401080	TERM	27	2	39	Cesarean section	2750	Male	47	35	2.65	AGA	389	265	46	234

IP250401081	TERM	23	2	40	Vaginal delivery	3200	Female	48	36.5	2.89	AGA	117	161	43	133
IP250401082	TERM	25	2	40	Vaginal delivery	4000	Male	49	36.5	3.40	AGA	278	262	35	197
IP250401083	TERM	28	2	38	Vaginal delivery	2950	Male	50	35.5	2.36	AGA	76	72	36	40
IP250401084	TERM	18	1	40	Vaginal delivery	2200	Female	49	34.5	1.87	SGA	393	247	36	176
IP250401085	TERM	25	1	37	Vaginal delivery	3000	Female	48	33	2.71	AGA	319	284	42	227
IP250401086	TERM	22	2	39	Cesarean section	3600	Female	47	36	3.47	AGA	296	210	57	151
IP250401087	TERM	20	1	39	Vaginal delivery	3800	Male	47.5	36	3.55	AGA	260	191	42	124
IP250401088	TERM	24	1	37	Cesarean section	2700	Female	45	34	2.96	AGA	249	162	39	122
IP250401089	TERM	32	1	37	Vaginal delivery	2810	Male	46	34	2.89	AGA	99	61	42	50
IP250401090	TERM	27	2	38	Cesarean section	2615	Male	50	33.5	2.09	AGA	243	230	63	160
IP250401091	TERM	23	1	40	Vaginal delivery	2000	Female	49	34.5	1.70	SGA	422	245	42	150
IP250401092	TERM	27	2	38	Vaginal delivery	3100	Male	48	33.5	2.80	AGA	51	176	107	110
IP250401093	TERM	24	2	37	Cesarean section	2100	Female	48	32	1.90	SGA	300	192	77	117
IP250401094	TERM	22	1	40	Vaginal delivery	2690	Female	49	34.5	2.29	SGA	285	258	49	191
IP250401095	TERM	22	1	41	Vaginal delivery	2650	Male	50	35	2.12	SGA	236	194	72	126
IP250401096	TERM	22	1	38	Vaginal delivery	2770	Male	46	34.5	2.85	AGA	274	158	39	98
IP250401097	TERM	21	1	39	Vaginal delivery	3500	Female	51	35	2.64	AGA	418	215	38	123
IP250401098	TERM	40	2	39	Vaginal delivery	3000	Male	48	33	2.71	AGA	151	166	29	117
IP250401099	TERM	21	1	39	Cesarean section	2700	Female	46.5	34	2.69	AGA	376	186	51	107
IP250401100	TERM	24	1	39	Cesarean section	2750	Female	47	34	2.65	AGA	202	195	35	157
IP250401101	TERM	25	1	40	Vaginal delivery	2300	Female	46.5	32.5	2.29	SGA	56	90	36	90
IP250401102	TERM	25	1	40	Vaginal delivery	3820	Female	54	36.5	2.43	AGA	249	334	42	265
IP250401103	TERM	20	1	40	Vaginal delivery	2500	Female	50	34.5	2.00	SGA	90	100	80	120
IP250401104	TERM	23	2	38	Cesarean section	3020	Male	50	35.5	2.42	AGA	455	528	44	380
IP250401105	TERM	25	1	40	Vaginal delivery	2830	Female	50.5	36.5	2.20	AGA	324	258	43	175
IP250401106	TERM	19	1	39	Vaginal delivery	3270	Male	47	36	3.15	AGA	397	205	33	153

IP250401107	TERM	25	1	40	Vaginal delivery	2620	Female	51	35.5	1.98	SGA	272	253	39	186
IP250401108	TERM	25	2	37	Cesarean section	2820	Male	46.5	34	2.80	AGA	301	257	51	201
IP250401109	TERM	20	1	38	Cesarean section	2900	Female	45.5	34.5	3.08	AGA	80	140	80	120
IP250401110	TERM	28	2	39	Vaginal delivery	3900	Female	48	36	3.53	AGA	423	272	88	182
IP250401111	TERM	29	2	40	Vaginal delivery	2400	Male	50	34.5	1.92	SGA	301	219	68	149
IP250401112	TERM	20	1	38	Cesarean section	2930	Female	48	34.5	2.65	AGA	119	122	41	167
IP250401113	TERM	20	1	38	Cesarean section	2900	Female	48	34.5	2.62	AGA	119	122	41	167
IP250401114	TERM	22	2	37	Vaginal delivery	2600	Female	45	33	2.85	AGA	89	131	40	165
IP250401115	TERM	25	2	38	Vaginal delivery	2400	Male	48	32.5	2.17	SGA	90	139	70	110
IP250401116	TERM	30	2	39	Vaginal delivery	2800	Male	45.5	34	2.97	AGA	251	250	42	130
IP250401117	TERM	22	2	37	Vaginal delivery	2800	Male	49	33	2.38	AGA	191	254	42	167
IP250401118	TERM	24	2	38	Cesarean section	2850	Male	47	34.5	2.75	AGA	363	260	40	200
IP250401119	TERM	23	2	40	Vaginal delivery	2750	Female	46.5	35.5	2.74	AGA	487	291	43	166
IP250401120	TERM	22	1	41	Vaginal delivery	3440	Male	49	37	2.92	AGA	278	262	35	197
IP250401121	TERM	26	2	39	Cesarean section	3460	Male	47	36	3.33	AGA	76	72	36	40
IP250401122	TERM	21	1	41	Vaginal delivery	2600	Female	50	35	2.08	SGA	393	247	36	176
IP250401123	TERM	22	1	38	Vaginal delivery	2350	Female	47	32.5	2.26	SGA	319	284	42	227
IP250401124	TERM	28	1	38	Vaginal delivery	3370	Female	48	34.5	3.05	AGA	296	210	57	151
IP250401125	TERM	24	1	38	Cesarean section	3200	Female	46	35.5	3.29	AGA	260	191	42	124
IP250401126	TERM	28	1	37	Vaginal delivery	2900	Male	48	33	2.62	AGA	249	162	39	122
IP250401127	TERM	25	2	38	Cesarean section	3460	Female	49.5	35.5	2.85	AGA	99	61	42	50
IP250401128	TERM	24	2	39	Vaginal delivery	2340	Female	48	33	2.12	SGA	243	230	63	160
IP250401129	TERM	19	1	37	Vaginal delivery	2310	Male	46	31	2.37	SGA	193	177	43	124
IP250401130	TERM	26	2	37	Cesarean section	3100	Female	48.5	33	2.72	AGA	283	244	45	142
IP250401131	TERM	24	2	37	Cesarean section	1770	Female	44	31	2.08	SGA	90	120	60	100
IP250401132	TERM	21	1	41	Cesarean section	2420	Female	48	34	2.19	SGA	200	201	44	115

IP250401133	TERM	30	2	38	Cesarean section	3090	Male	48	34.5	2.79	AGA	228	248	40	181
IP250401134	TERM	24	1	39	Vaginal delivery	2990	Female	47	35	2.88	AGA	220	265	45	140
IP250401135	TERM	21	3	39	Vaginal delivery	2200	Female	47	33	2.12	SGA	145	189	52	102
IP250401136	TERM	28	2	40	Vaginal delivery	2800	Female	46	35.5	2.88	AGA	278	293	36	99
IP250401137	TERM	27	3	39	Vaginal delivery	2980	Male	45	34	3.27	AGA	174	275	43	139
IP250401138	TERM	21	2	38	Vaginal delivery	2000	Female	46	31.5	2.05	SGA	140	195	44	133
IP250401139	TERM	30	2	38	Vaginal delivery	3560	Male	48	35.5	3.22	AGA	82	106	36	69
IP250401140	TERM	19	1	41	Vaginal delivery	2100	Female	49	35	1.78	SGA	262	307	45	99
IP250401141	TERM	22	1	40	Vaginal delivery	2300	Male	49	34.5	1.95	SGA	264	253	41	212
IP250401142	TERM	20	1	40	Vaginal delivery	3200	Female	47	36.5	3.08	AGA	110	110	55	130
IP250401143	TERM	22	2	39	Vaginal delivery	3110	Male	49	35	2.64	AGA	182	262	42	154
IP250401144	TERM	24	3	39	Vaginal delivery	2000	Male	51	35	1.51	SGA	105	106	41	71
IP250401145	TERM	28	2	39	Cesarean section	2350	Male	47	32	2.26	SGA	195	285	44	216
IP250401146	TERM	28	2	40	Vaginal delivery	3400	Female	46.5	36.5	3.38	AGA	409	374	58	228
IP250401147	TERM	27	1	38	Vaginal delivery	3200	Male	47	34.5	3.08	AGA	160	83	35	160
IP250401148	TERM	23	2	39	Vaginal delivery	2300	Female	47	32	2.22	SGA	178	140	43	56
IP250401149	TERM	35	2	37	Vaginal delivery	2900	Female	46	33	2.98	AGA	335	230	42	140
IP250401150	TERM	27	3	41	Vaginal delivery	3900	Male	48	37	3.53	AGA	119	122	41	167
IP250401151	TERM	31	3	38	Vaginal delivery	1500	Male	44	31.5	1.76	SGA	119	122	41	167
IP250401152	TERM	28	2	37	Vaginal delivery	2400	Male	50	32	1.92	SGA	89	131	40	165
IP250401153	TERM	20	1	37	Vaginal delivery	2600	Male	45	32	2.85	AGA	76	72	36	40
IP250401154	TERM	27	2	38	Vaginal delivery	2100	Male	48	31.5	1.90	SGA	393	247	36	176
IP250401155	TERM	27	2	39	Cesarean section	2000	Male	47	32	1.93	SGA	220	265	45	140

Registration ID	GROUP	Age of the patient	GRAVIDA	POG	Mode of delivery	Birth weight	Sex of the baby	head circumfre	length	pI	aga/sga	percentile	LDL	Serum total chol	Serum HDL	Serum triglycerides
IP250401156	PRETERM	23	2	28	Vaginal delivery	800	Female	23.2	35	1.87	SGA	<10th percentile	237	214	42	145
IP250401157	PRETERM	28	2	29	Vaginal delivery	800	Female	21.4	35	1.87	SGA	<10th percentile	334	107	35	85
IP250401158	PRETERM	25	2	29	Cesarean section	900	Female	22	36	1.93	SGA	<10th percentile	388	275	45	209
IP250401159	PRETERM	22	1	36	Cesarean section	1100	Female	26.4	40	1.72	SGA	<10th percentile	333	200	26	150
IP250401160	PRETERM	25	1	36	Vaginal delivery	1170	Female	26.1	39	1.97	SGA	<10th percentile	420	283	64	225
IP250401161	PRETERM	27	1	30	Vaginal delivery	1200	Female	26.1	39	2.02	AGA	10th-50th percentile	336	203	42	91
IP250401162	PRETERM	19	1	29	Vaginal delivery	1200	Female	29	45	1.32	AGA	10th-50th percentile	351	236	32	183
IP250401163	PRETERM	21	1	28	Vaginal delivery	1216	Female	23.8	34	3.09	AGA	50th-90th percentile	176	196	36	127
IP250401164	PRETERM	21	2	28	Vaginal delivery	1216	Female	23.8	34	3.09	AGA	50th-90th percentile	122	170	41	115
IP250401165	PRETERM	26	1	29	Vaginal delivery	1300	Female	29.1	45	1.43	AGA	50th-90th percentile	165	268	40	202
IP250401166	PRETERM	22	1	29	Vaginal delivery	1300	Female	27.7	42	1.75	AGA	50th-90th percentile	259	311	55	204
IP250401167	PRETERM	20	1	32	Vaginal delivery	1300	Female	27.7	42	1.75	SGA	<10th percentile	334	107	35	85
IP250401168	PRETERM	28	1	32	Cesarean section	1300	Female	26.7	40	2.03	SGA	<10th percentile	308	316	42	257
IP250401169	PRETERM	24	1	28	Cesarean section	1310	Male	27.2	41	1.90	AGA	50th-90th percentile	388	275	45	209
IP250401170	PRETERM	25	2	31	Cesarean section	1340	Male	29.6	46	1.38	SGA	<10th percentile	226	234	36	173
IP250401171	PRETERM	20	2	31	Vaginal delivery	1350	Male	27.7	42	1.82	SGA	<10th percentile	388	275	45	209
IP250401172	PRETERM	23	1	36	Vaginal delivery	1360	Female	25.4	37	2.68	SGA	<10th percentile	498	340	45	349
IP250401173	PRETERM	24	1	36	Cesarean section	1360	Female	25.4	37	2.68	SGA	<10th percentile	145	240	67	159
IP250401174	PRETERM	26	2	31	Vaginal delivery	1500	Male	29.3	45	1.65	AGA	10th-50th percentile	237	214	42	145
IP250401175	PRETERM	21	1	36	Vaginal delivery	1500	Male	27	40	2.34	SGA	<10th percentile	176	196	36	127
IP250401176	PRETERM	20	1	36	Vaginal delivery	1590	Male	27.1	40	2.48	SGA	<10th percentile	122	170	41	115
IP250401177	PRETERM	20	1	32	Cesarean section	1670	Male	30.5	47	1.61	AGA	10th-50th percentile	308	316	42	257

IP250401178	PRETERM	25	2	32	Vaginal delivery	1700	Male	25.9	37	3.36	AGA	10th-50th percentile	351	236	32	183
IP250401179	PRETERM	32	2	33	Cesarean section	1720	Female	28.2	42	2.32	AGA	10th-50th percentile	237	214	42	145
IP250401180	PRETERM	22	2	33	Cesarean section	1720	Female	28.2	42	2.32	AGA	10th-50th percentile	363	260	40	200
IP250401181	PRETERM	25	1	32	Vaginal delivery	1740	Male	29.6	45	1.91	AGA	10th-50th percentile	122	170	41	115
IP250401182	PRETERM	27	2	32	Vaginal delivery	1740	Male	28.7	43	2.19	AGA	10th-50th percentile	336	203	42	91
IP250401183	PRETERM	26	2	31	Cesarean section	1790	Female	27.3	40	2.80	AGA	50th-90th percentile	267	268	55	192
IP250401184	PRETERM	30	1	34	Vaginal delivery	1800	Female	26.5	42	2.43	SGA	<10th percentile	231	204	44	166
IP250401185	PRETERM	20	1	34	Vaginal delivery	1800	Female	26.5	43	2.26	SGA	<10th percentile	498	340	45	349
IP250401186	PRETERM	24	2	34	Vaginal delivery	1850	Female	28.8	43	2.33	AGA	10th-50th percentile	321	340	42	116
IP250401187	PRETERM	22	2	34	Vaginal delivery	1900	Male	29.8	46	1.95	SGA	<10th percentile	167	112	36	82
IP250401188	PRETERM	23	2	34	Vaginal delivery	1900	Male	29.3	44	2.23	SGA	<10th percentile	165	268	40	202
IP250401189	PRETERM	24	1	35	Cesarean section	1970	Male	30.4	47	1.90	SGA	<10th percentile	165	268	40	202
IP250401190	PRETERM	23	2	35	Vaginal delivery	1970	Male	26.2	47	1.90	SGA	<10th percentile	282	238	45	166
IP250401191	PRETERM	24	2	32	Vaginal delivery	1980	Female	29.4	44	2.32	AGA	50th-90th percentile	191	254	42	167
IP250401192	PRETERM	22	2	32	Vaginal delivery	1980	Female	28.5	42	2.67	AGA	50th-90th percentile	231	204	44	166
IP250401193	PRETERM	21	1	32	Vaginal delivery	1990	Male	30.4	46	2.04	AGA	50th-90th percentile	122	170	41	115
IP250401194	PRETERM	26	2	32	Vaginal delivery	1995	Male	27.6	40	3.12	AGA	50th-90th percentile	282	238	45	166
IP250401195	PRETERM	30	1	36	Vaginal delivery	2000	Male	29	46	2.05	SGA	<10th percentile	282	238	45	166
IP250401196	PRETERM	30	2	34	Vaginal delivery	2000	Male	27.6	40	3.13	AGA	10th-50th percentile	420	283	64	225
IP250401197	PRETERM	29	2	36	Vaginal delivery	2000	Female	30.9	47	1.93	SGA	<10th percentile	237	214	42	145
IP250401198	PRETERM	24	2	34	Vaginal delivery	2000	Male	29	43	2.52	AGA	10th-50th percentile	267	268	55	192
IP250401199	PRETERM	23	1	36	Cesarean section	2080	Male	29.6	48	1.88	SGA	<10th percentile	145	240	67	159
IP250401200	PRETERM	27	2	35	Vaginal delivery	2100	Male	30.1	45	2.30	SGA	<10th percentile	321	340	42	116
IP250401201	PRETERM	30	2	35	Vaginal delivery	2100	Male	27.8	45	2.30	SGA	<10th percentile	182	174	47	133
IP250401202	PRETERM	35	2	33	Vaginal delivery	2100	Male	30.5	46	2.16	AGA	50th-90th percentile	282	238	45	166
IP250401203	PRETERM	31	2	33	Vaginal delivery	2100	Male	30.1	45	2.30	AGA	50th-90th percentile	259	311	55	204

IP250401204	PRETERM	28	2	33	Vaginal delivery	2100	Male	30.1	45	2.30	AGA	50th-90th percentile	334	107	35	85
IP250401205	PRETERM	19	2	32	Cesarean section	2100	Male	28.2	41	3.05	AGA	50th-90th percentile	176	196	36	127
IP250401206	PRETERM	25	2	35	Vaginal delivery	2100	Male	31.5	48	1.90	SGA	<10th percentile	351	236	32	183
IP250401207	PRETERM	30	2	36	Vaginal delivery	2100	Male	31	49	1.78	SGA	<10th percentile	165	268	40	202
IP250401208	PRETERM	30	1	36	Vaginal delivery	2100	Male	29.6	48	1.90	SGA	<10th percentile	282	238	45	166
IP250401209	PRETERM	27	2	35	Vaginal delivery	2100	Female	28.7	42	2.83	AGA	10th-50th percentile	336	203	42	91
IP250401210	PRETERM	24	2	33	Vaginal delivery	2100	Male	27.8	40	3.28	AGA	50th-90th percentile	191	254	42	167
IP250401211	PRETERM	23	1	32	Cesarean section	2100	Male	29.6	44	2.47	AGA	50th-90th percentile	145	240	67	159
IP250401212	PRETERM	19	1	35	Vaginal delivery	2100	Male	29.1	46	2.16	SGA	<10th percentile	397	327	49	218
IP250401213	PRETERM	21	2	34	Vaginal delivery	2150	Female	30.1	45	2.36	AGA	50th-90th percentile	122	170	41	115
IP250401214	PRETERM	30	1	36	Vaginal delivery	2150	Female	29.2	48	1.94	SGA	<10th percentile	167	112	36	82
IP250401215	PRETERM	30	2	34	Vaginal delivery	2150	Female	30.1	45	2.36	AGA	50th-90th percentile	182	174	47	133
IP250401216	PRETERM	30	2	35	Vaginal delivery	2180	Female	32.1	49	1.85	AGA	10th-50th percentile	363	260	40	200
IP250401217	PRETERM	22	1	36	Cesarean section	2190	Female	29.7	46	2.25	SGA	<10th percentile	333	200	26	150
IP250401218	PRETERM	22	1	36	Vaginal delivery	2190	Female	29.7	45	2.40	SGA	<10th percentile	259	311	55	204
IP250401219	PRETERM	20	1	36	Vaginal delivery	2190	Female	29.7	48	1.98	SGA	<10th percentile	334	107	35	85
IP250401220	PRETERM	26	1	36	Vaginal delivery	2200	Female	32.1	49	1.87	SGA	<10th percentile	191	254	42	167
IP250401221	PRETERM	22	2	36	Vaginal delivery	2200	Female	31.6	48	1.99	SGA	<10th percentile	231	204	44	166
IP250401222	PRETERM	21	1	36	Vaginal delivery	2220	Male	29.3	48	2.01	SGA	<10th percentile	122	170	41	115
IP250401223	PRETERM	22	2	36	Vaginal delivery	2220	Male	29.3	49	1.89	SGA	<10th percentile	167	112	36	82
IP250401224	PRETERM	26	1	35	Vaginal delivery	2230	Male	29.8	44	2.62	AGA	10th-50th percentile	191	254	42	167
IP250401225	PRETERM	19	1	35	Vaginal delivery	2240	Male	30.2	45	2.46	AGA	10th-50th percentile	397	327	49	218
IP250401226	PRETERM	28	2	36	Vaginal delivery	2240	Male	28.8	47	2.16	SGA	<10th percentile	182	174	47	133
IP250401227	PRETERM	32	2	35	Cesarean section	2240	Male	30.2	45	2.46	AGA	10th-50th percentile	237	214	42	145
IP250401228	PRETERM	22	2	36	Cesarean section	2240	Male	28.8	47	2.16	SGA	<10th percentile	363	260	40	200
IP250401229	PRETERM	20	2	36	Vaginal delivery	2300	Male	28.9	47	2.22	SGA	<10th percentile	237	214	42	145

IP250401230	PRETERM	26	2	36	Vaginal delivery	2300	Male	28	49	1.95	SGA	<10th percentile	282	238	45	166
IP250401231	PRETERM	27	1	35	Vaginal delivery	2300	Male	30.3	45	2.52	AGA	10th-50th percentile	237	214	42	145
IP250401232	PRETERM	27	1	36	Vaginal delivery	2300	Female	27.1	38	4.19	AGA	10th-50th percentile	336	203	42	91
IP250401233	PRETERM	30	1	34	Vaginal delivery	2300	Male	32.7	50	1.84	AGA	50th-90th percentile	167	112	36	82
IP250401234	PRETERM	20	1	34	Vaginal delivery	2300	Male	30.8	46	2.36	AGA	50th-90th percentile	122	170	41	115
IP250401235	PRETERM	26	2	35	Cesarean section	2300	Female	30.3	45	2.52	AGA	10th-50th percentile	267	268	55	192
IP250401236	PRETERM	25	2	35	Cesarean section	2315	Male	30.3	45	2.54	AGA	10th-50th percentile	226	234	36	173
IP250401237	PRETERM	24	1	34	Vaginal delivery	2330	Male	31.3	47	2.24	AGA	50th-90th percentile	237	214	42	145
IP250401238	PRETERM	20	1	34	Cesarean section	2330	Male	30.3	45	2.56	AGA	50th-90th percentile	308	316	42	257
IP250401239	PRETERM	25	1	34	Cesarean section	2360	Male	31.3	47	2.27	AGA	50th-90th percentile	333	200	26	150
IP250401240	PRETERM	20	2	35	Vaginal delivery	2360	Female	30.4	45	2.59	AGA	10th-50th percentile	388	275	45	209
IP250401241	PRETERM	20	2	35	Vaginal delivery	2360	Male	30.4	45	2.59	AGA	10th-50th percentile	237	214	42	145
IP250401242	PRETERM	23	2	34	Vaginal delivery	2370	Male	29.9	44	2.78	AGA	50th-90th percentile	237	214	42	145
IP250401243	PRETERM	25	1	35	Vaginal delivery	2400	Female	30.9	46	2.47	AGA	50th-90th percentile	420	283	64	225
IP250401244	PRETERM	25	2	35	Vaginal delivery	2400	Male	30	44	2.82	AGA	10th-50th percentile	237	214	42	145
IP250401245	PRETERM	25	3	35	Cesarean section	2400	Female	31.8	48	2.17	AGA	50th-90th percentile	388	275	45	209
IP250401246	PRETERM	33	3	33	Vaginal delivery	2400	Male	31.8	48	2.17	AGA	50th-90th percentile	308	316	42	257
IP250401247	PRETERM	24	1	35	Cesarean section	2400	Female	31.4	47	2.31	AGA	50th-90th percentile	388	275	45	209
IP250401248	PRETERM	22	1	35	Cesarean section	2400	Male	30.4	45	2.63	AGA	10th-50th percentile	176	196	36	127
IP250401249	PRETERM	25	1	36	Vaginal delivery	2400	Female	30.4	45	2.63	AGA	10th-50th percentile	122	170	41	115
IP250401250	PRETERM	28	3	33	Cesarean section	2400	Male	30.4	45	2.63	AGA	50th-90th percentile	363	260	40	200
IP250401251	PRETERM	28	3	35	Cesarean section	2410	Male	31.8	48	2.18	AGA	10th-50th percentile	363	260	40	200
IP250401252	PRETERM	26	3	35	Cesarean section	2415	Male	30.4	45	2.65	AGA	10th-50th percentile	267	268	55	192
IP250401253	PRETERM	23	3	36	Vaginal delivery	2430	Male	30.9	46	2.50	AGA	10th-50th percentile	167	112	36	82
IP250401254	PRETERM	23	3	36	Vaginal delivery	2430	Male	30.9	46	2.50	AGA	10th-50th percentile	282	238	45	166
IP250401255	PRETERM	25	1	36	Vaginal delivery	2430	Male	30.5	45	2.67	AGA	10th-50th percentile	420	283	64	225

IP250401256	PRETERM	25	1	35	Vaginal delivery	2450	Male	31	46	2.52	AGA	10th-50th percentile	363	260	40	200
IP250401257	PRETERM	30	3	35	Cesarean section	2450	Female	30.5	45	2.69	AGA	50th-90th percentile	397	327	49	218
IP250401258	PRETERM	31	1	36	Vaginal delivery	2450	Male	29.6	43	3.08	AGA	10th-50th percentile	226	234	36	173
IP250401259	PRETERM	24	3	35	Vaginal delivery	2450	Male	30	44	2.88	AGA	10th-50th percentile	321	340	42	116
IP250401260	PRETERM	26	3	36	Vaginal delivery	2450	Female	29.6	43	3.08	AGA	10th-50th percentile	388	275	45	209
IP250401261	PRETERM	23	3	36	Vaginal delivery	2450	Female	28.2	40	3.83	AGA	10th-50th percentile	167	112	36	82
IP250401262	PRETERM	26	3	35	Vaginal delivery	2490	Female	31	46	2.56	AGA	50th-90th percentile	237	214	42	145
IP250401263	PRETERM	20	1	36	Cesarean section	2500	Female	31.5	47	2.41	AGA	10th-50th percentile	176	196	36	127
IP250401264	PRETERM	23	1	35	Vaginal delivery	2500	Female	30.1	44	2.93	AGA	50th-90th percentile	498	340	45	349
IP250401265	PRETERM	24	1	35	Cesarean section	2500	Female	32.9	50	2.00	AGA	50th-90th percentile	145	240	67	159
IP250401266	PRETERM	30	3	36	Cesarean section	2500	Female	32.9	50	2.00	AGA	10th-50th percentile	397	327	49	218
IP250401267	PRETERM	24	3	34	Vaginal delivery	2500	Male	31.5	47	2.41	AGA	50th-90th percentile	267	268	55	192
IP250401268	PRETERM	22	3	35	Vaginal delivery	2500	Female	30.5	45	2.74	AGA	50th-90th percentile	420	283	64	225
IP250401269	PRETERM	19	1	35	Vaginal delivery	2500	Female	30.5	45	2.74	AGA	50th-90th percentile	351	236	32	183
IP250401270	PRETERM	24	1	35	Cesarean section	2500	Female	33.9	52	1.78	AGA	50th-90th percentile	165	268	40	202
IP250401271	PRETERM	26	3	34	Cesarean section	2500	Male	32.9	50	2.00	AGA	50th-90th percentile	308	316	42	257
IP250401272	PRETERM	24	1	36	Vaginal delivery	2500	Female	30.1	44	2.93	AGA	10th-50th percentile	237	214	42	145
IP250401273	PRETERM	30	3	36	Vaginal delivery	2500	Female	29.2	42	3.37	AGA	10th-50th percentile	165	268	40	202
IP250401274	PRETERM	20	1	35	Cesarean section	2510	Female	31	46	2.58	AGA	50th-90th percentile	176	196	36	127
IP250401275	PRETERM	33	2	35	Vaginal delivery	2510	Female	29.2	42	3.39	AGA	50th-90th percentile	308	316	42	257
IP250401276	PRETERM	30	1	35	Vaginal delivery	2515	Female	31.5	47	2.42	AGA	50th-90th percentile	231	204	44	166
IP250401277	PRETERM	28	1	35	Cesarean section	2515	Female	30.6	45	2.76	AGA	50th-90th percentile	308	316	42	257
IP250401278	PRETERM	27	2	35	Vaginal delivery	2560	Male	31.1	46	2.63	AGA	50th-90th percentile	321	340	42	116
IP250401279	PRETERM	25	2	35	Vaginal delivery	2560	Male	30.6	45	2.81	AGA	50th-90th percentile	237	214	42	145
IP250401280	PRETERM	22	2	35	Vaginal delivery	2560	Female	29.7	43	3.22	AGA	50th-90th percentile	420	283	64	225
IP250401281	PRETERM	26	2	35	Vaginal delivery	2590	Female	32.1	48	2.34	AGA	50th-90th percentile	388	275	45	209

IP250401282	PRETERM	31	1	35	Vaginal delivery	2600	Female	32.5	49	2.21	AGA	50th-90th percentile	226	234	36	173
IP250401283	PRETERM	26	2	36	Cesarean section	2610	Male	33.5	51	1.97	AGA	10th-50th percentile	267	268	55	192
IP250401284	PRETERM	26	2	36	Cesarean section	2610	Male	31.6	47	2.51	AGA	10th-50th percentile	267	268	55	192
IP250401285	PRETERM	26	1	36	Vaginal delivery	2650	Female	31.7	47	2.55	AGA	50th-90th percentile	165	268	40	202
IP250401286	PRETERM	23	2	36	Vaginal delivery	2650	Male	30.7	45	2.91	AGA	10th-50th percentile	165	268	40	202
IP250401287	PRETERM	20	1	36	Vaginal delivery	2690	Female	32.2	48	2.43	AGA	50th-90th percentile	498	340	45	349
IP250401288	PRETERM	25	1	36	Cesarean section	2690	Female	30.8	45	2.95	AGA	50th-90th percentile	333	200	26	150
IP250401289	PRETERM	27	2	36	Vaginal delivery	2700	Male	34.1	52	1.92	AGA	10th-50th percentile	167	112	36	82
IP250401290	PRETERM	26	2	36	Cesarean section	2700	Male	33.1	50	2.16	AGA	10th-50th percentile	267	268	55	192
IP250401291	PRETERM	30	2	36	Vaginal delivery	2700	Male	31.7	47	2.60	AGA	10th-50th percentile	363	260	40	200
IP250401292	PRETERM	19	4	36	Cesarean section	2720	Female	30.8	45	2.98	AGA	50th-90th percentile	176	196	36	127
IP250401293	PRETERM	26	4	36	Cesarean section	2800	Female	33.2	50	2.24	AGA	50th-90th percentile	308	316	42	257
IP250401294	PRETERM	28	4	36	Vaginal delivery	2800	Female	30.5	44	3.29	AGA	50th-90th percentile	182	174	47	133
IP250401295	PRETERM	24	4	36	Vaginal delivery	2800	Female	30	43	3.52	AGA	50th-90th percentile	237	214	42	145
IP250401296	PRETERM	31	4	36	Vaginal delivery	2880	Female	31.5	46	2.96	AGA	50th-90th percentile	259	311	55	204
IP250401297	PRETERM	35	4	35	Vaginal delivery	2900	Female	32	47	2.79	AGA	50th-90th percentile	282	238	45	166
IP250401298	PRETERM	24	4	36	Vaginal delivery	2910	Male	32.9	49	2.47	AGA	50th-90th percentile	237	214	42	145
IP250401299	PRETERM	25	1	36	Vaginal delivery	2910	Male	32	47	2.80	AGA	50th-90th percentile	420	283	64	225
IP250401300	PRETERM	27	4	36	Vaginal delivery	2920	Male	32.4	48	2.64	AGA	50th-90th percentile	167	112	36	82
IP250401301	PRETERM	22	1	36	Cesarean section	3000	Male	35.4	54	1.91	AGA	50th-90th percentile	176	196	36	127